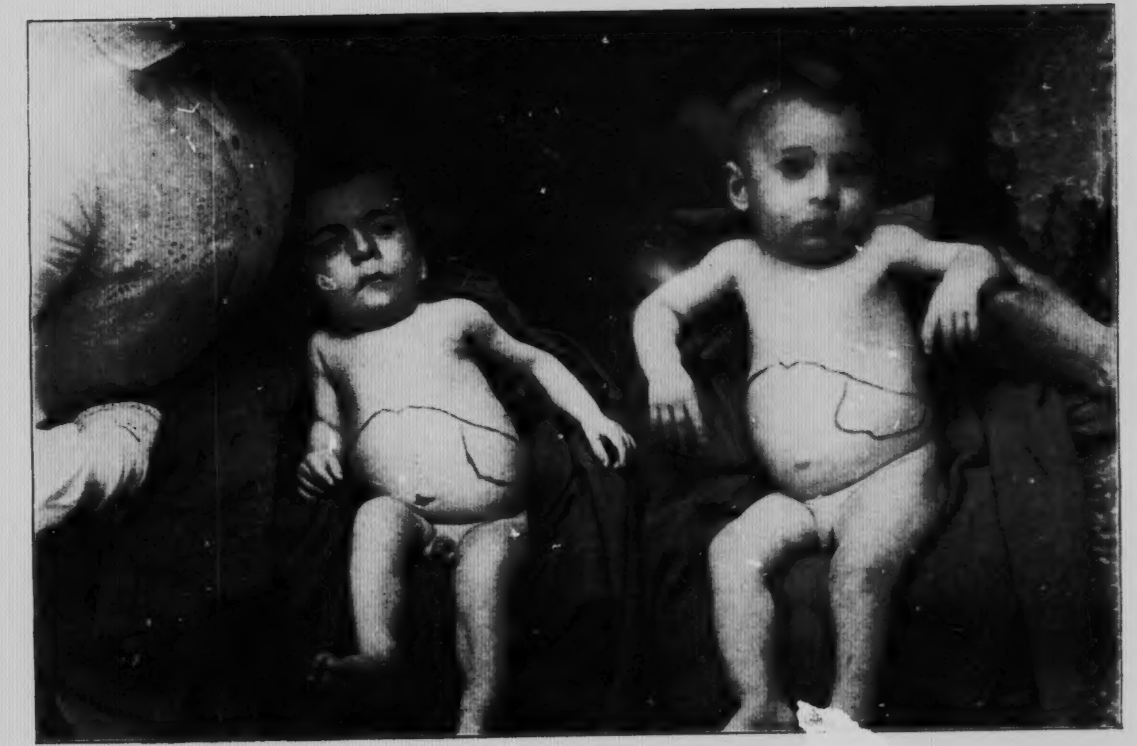
LECTURES ON DISEASES OF CHILDREN



SPLENIC ANÆMIA IN TWINS.

LECTURES

ON

DISEASES OF CHILDREN

BY

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TO

DR. JOHN THOMSON,

MY FIRST TEACHER IN DISEASES OF CHILDREN,

I DEDICATE THIS BOOK

IN RECOGNITION OF HIS EMINENCE AS A

WORKER IN THIS FIELD

AND IN GRATITUDE FOR A LONG FRIENDSHIP

PREFACE TO THE THIRD EDITION

In the present edition the whole book has been revised, and six new lectures (Nos. VII., XVI., XVII., XX., XXIV., and XXVI.), and several new illustrations added. The general scope and plan of the work, however, remain unaltered.

R. H.

Queen Anne Street, W.
January, 1913.

PREFACE TO THE FIRST EDITION

The lectures in this book were delivered in the form of a systematic course at the London Hospital two years ago, and first published serially in the *Clinical Journal*. I have gathered them together at the request of many of those who listened to them, and who have been kind enough to express a wish to have them in a more convenient form for reference.

The aim and scope of the book are sufficiently indicated in the opening paragraphs of the first lecture, but I would only say here that it is not intended in any sense to be an exhaustive treatise on the diseases of childhood, nor to compete with the many admirable text-books of that subject already in existence. My object has simply been to describe those common diseases of children which are

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not usually dealt with in systematic lectures on medicine, and which are apt to receive but little attention in ordinary ward teaching. The subject has been approached from a purely clinical standpoint, and with but little reference to problems of pathology; on the other hand, questions of treatment have been dealt with in some detail. The fact that the lectures were delivered from only a few notes and have been merely corrected from the stenographer's report must serve as an excuse for the rather colloquial character of the style in places, and for not infrequent repetitions.

The illustrations are, with one or two exceptions, original, and have been reproduced from photographs of my own cases by lantern-slides of which the lectures were illustrated.

I am indebted to my friend Mr. Robert Tanner for the care he has bestowed on the revision of the proof-sheets, and must express also my great obligation to Dr. Eliot Creasy, editor of the *Clinical Journal*, for his permission to reproduce the lectures in book form. The index is the work of Mr. Hewitt, of the Royal College of Surgeons.

R. H.

July, 1904.

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LECTURES ON DISEASES OF CHILDREN

LECTURE I

THE CLINICAL EXAMINATION OF SICK CHILDREN

GENTLEMEN,—It will be generally admitted, I think, that the study of diseases of children, which will occupy us in these lectures, is one which is apt to be neglected in general hospitals. One reason for this probably is that adult patients are so numerous, and their claims on the staff are so pressing, that there is very little time left for special teaching in the children's wards. And also, I think, it has its explanation in this further fact, that the teaching of general hospitals is, and necessarily must be, directed specially to the requirements of qualifying examinations. Now it is—in my judgment, at least—unfortunate, though I can hardly expect to carry your sympathies in this matter, that there is no demand made at the ordinary qualifying examinations for a special knowledge of the diseases of children, the consequence of which is that many men when they qualify know almost nothing of many of the commonest ailments of infant life. On going out into practice you are not much better informed in many cases than the mother or the nurse as to the diseases which you have got to deal with. And yet you will find that in any large general practice children make up a great part, perhaps the greater part, of your patients, and the seriousness of their diseases will be brought home to you by a study of the death-rate, for whereas the adult mortality has gone down in the last fifty years by about 4 per 1,000, the infantile mortality is as high as ever it was; and, indeed, of every hundred children born fifteen never see their first birthday. Now, gentlemen, that is not creditable to our profession, and although I admit that much of it is due to social and economic causes for which you and I are not in any way directly responsible, yet I cannot doubt that, if all practitioners had the opportunity as students of becoming familiar with the treatment of the common diseases of infancy, the result would be seen in an appreciable diminution of our infant mortality.

I should be sorry, however, if you were to embark upon this course of study merely from such utilitarian motives as those I have put forward, because I can promise you that on scientific grounds alone, alike pathological and therapeutic, there is no subject which will better repay your attention than the study of the diseases of children. You will find that pathological processes in children tend, if I may use the term, to be pure bred; they are not hybrids produced by a blending of different morbid factors. Disease in children also runs a less complicated course than it does in the adult, and tends to exhibit pathological processes in their simplest forms.

As regards therapeutics, you will find treatment in children eminently satisfactory. I am bound to confess that when you study the results of the treatment of disease in adults in large hospitals such as this, you are tempted to form a rather low opinion of the potentialities of medical therapeutics. Frequent failure is inevitable; it depends largely on the circumstances in which our patients in this part of London are placed. But with children it is not so; they respond to treatment in a most surprising and gratifying fashion. One reason is, no doubt, that you can so easily modify your patient's surroundings. A child has not got to work; you can take him away from school for an indefinite number of weeks, and no one will object except the schoolmaster or the attendance officer. Moreover, in the case of children you are fighting with Nature instead of against her. In the case of most of our adult patients they are going down the hill or are about to go down it; but children are going up the hill, and they have great reparative power to fall back upon, and hence it is that the treatment of their diseases is so encouraging. All the more reason, therefore, that you should know how to treat these diseases, for you will often find when you go into practice that the life of the child is literally in your hands, and that a mistake at a critical moment may be really a matter of life and death.

Now, it has always been the boast of this hospital that whatever it may do or leave undone, at all events it offers students unrivalled opportunity of fitting themselves for practice But although this is true, and although we treat here a much larger number of sick children than any other general hospital, there has not hitherto been given here, any more than at any of the other Metropolitan Schools, systematic instruction in the special diseases of childhood. That gap in your training I should consider it a privilege to be able to fill, and I have therefore invited you to meet me here, by the consent and with the encouragement of my colleagues, in order that we may consider together, briefly and practically, the nature and treatment of those diseases which are peculiar to early life.

I may now turn, gentlemen, to the subject of the first lecture of this course—the **methods of examining children** clinically. I must ask you to bear with me if you find this not extremely interesting, but it is an unavoidable preliminary, for the methods of examining and handling a sick child do not come to you by intuition. It was pointed out by Dr. Charles West, who was one of the pioneers in this subject, that the man who starts for the first time to study the diseases of children is like a traveller in a foreign country; he hears a strange language spoken which he does not understand. At all events, if the language is not absolutely strange, it is spoken with a foreign accent, for the physical signs of disease are often different in children to what they are in grown-up people, and so you have to devote special attention to their interpretation.

Begin with the **history.** The child, of course, will be unable to tell you anything, or but very little, and you have to fall back upon the information supplied by the mother or the nurse for a history of the illness. These are apt to be loquacious persons, but it is always worth while to listen patiently to all they have to say, for there is nobody in such a favourable position to observe small changes in health of the child as those who are constantly attending to him. Listen attentively, then, to what the mother or the nurse has to say as to the mode of onset of the illness, and the symptoms which the child exhibits.

With what are called 'subjective symptoms' in children you are not in any way troubled, because there are none. Pediatrics is like veterinary work in this, that the patient is unable to give you an account of his sufferings, and you are thrown back entirely on your own observation, which, of course, makes the necessity for careful examination all the greater. With regard to the history, I would say that nutrition and heredity play such a large part in the diseases of children that you must always make a special investigation into these points. You must ask, as regards the family history, what other children there have been, how many of those are living, and how many, if any, are dead, and the ages at which they died; you must inquire also about the health of the mother during pregnancy, whether the child was carried to term or not, and as to the nature of the labour by which the child was ushered into the world. All these points have a direct bearing on the conclusions which you will draw as to the kind of heredity with which your patient is burdened. You will further inquire with regard to nutrition, noting the exact mode in which the child has been fed, as well as the particular food or foods which are being given now.

Answers to these questions will clear the ground, and will prepare you for proceeding to your **physical examination.** I need hardly emphasize here the importance of gentleness and tact. These things cannot be taught—they come naturally to some people—but everybody can acquire some degree of them by care and with opportunities for practice. Children are difficult to handle: they are easily frightened, and they distrust the presence of a stranger, and these things add to the difficulties which you will encounter. The methods which you adopt in your examination of children are the usual methods of inspection, palpation, percussion, and auscultation. These have not the same relative values.

however, as in the case of adults. Inspection is immeasurably the most important method in a child. The next in importance is palpation, next auscultation ; percussion we will leave till last.

You have already, of course, gleaned much information by casual observation whilst you have been talking to the mother and getting the history. During that time the child has become accustomed to your presence. You will have noticed whether he looks seriously ill, whether he is interested in his toys or surroundings, and whether he presents any obvious signs of disease. Having done that, you have to proceed to more exact **inspection.** For that purpose it is important to have the child completely stripped. It is well that this should be done before you come into the room ; it is bad to get a baby to associate you in its mind with the taking off of its clothes. The next piece of advice I would give you is never to look a baby in the face. That, of course, does not apply to grown-up persons ; we feel inclined to distrust a man who does not look us in the face, but the baby distrusts the man who does. There is no surer way of making a baby cry, and therefore of making it far more difficult to examine, than by staring it in the face.

You should begin your inspection by noting the child's **expression.** Much has been written about the facies of disease, and you will find mysterious lines described as coming out on the child's face when it is ill. I do not think any good purpose would be served if I attempted to describe them to you. That is a matter which you must pick up as you have opportunity here, where you will be able to see the facies of all sorts of diseases in infants, and you will get familiar by-and-by with the facies of abdominal disease, with the facies of acute respiratory disease, of cerebral disease, and so on. The importance of this subject, however, justifies all that has been written about it, because the face of the child is a mirror in a way in which that of a grown-up person is not. It is a clean sheet upon which disease can write ; it is not furrowed over with the lines of care and anxiety, nor does it show those changes so often to be seen in adults as a result of debauchery and bad living.

Moreover, the signs of disease in a child are not masked by the emotions nor suppressed by the will.

Now, having noted these things, you cast your eye down over the child, and you will observe certain **peculiarities of configuration** which are normal, but which the child's mother may think are abnormal. One of these is that the child has a relatively large abdomen. You will not be long in practice before babies are brought to you by anxious mothers with the complaint that 'the stomach is getting large.' I do not say that these mothers are not occasionally right, and that the abdomen is not pathologically enlarged sometimes, but it is well to note that a child's abdomen is naturally prominent, the main reason for which is that the liver is relatively bigger and occupies a considerable space in the abdomen.

Another peculiarity is that the chest is very round, and it is only after some time that it acquires the oval shape which is seen in a well-formed adult. There are certain consequences of this round-shaped chest in children which I shall have to point out to you when we come to study respiratory disease. Another point to note is the relatively large head of the child compared with the trunk. Here, again, anxious mothers come to you complaining that the child's head is getting large, and possibly the neighbours have been trying to make her believe that the baby has got 'water on the brain.' Remember in such an event that a child is about three years of age before the head and chest have the same relative size as in the adult. Of course, this is easily understood when you remember that during those years the child's brain is growing rapidly.

Other points to notice are the character of the respiratory movements—whether they are rapid, regular, easy, or laboured. You will also notice whether there is any head retraction. You will observe the position of the limbs—whether they are held in an easy and natural posture, or kept rigid, as they are apt to be when the seat of painful disease, such as scurvy. Look also for the signs of rickets, which you will quickly learn to recognise when we come to speak of that disease. You will, at the same time, notice the presence or absence of rashes, which are so important a diagnostic sign in the diseases of infancy.

Having run your eye rapidly over the child in this way, you come to the next method of examination—namely, **palpation.** The advice used to be given by an old teacher of mine that you should always 'paw your babies.' That was very sound advice. But I would remind you of the importance of a warm hand in doing so. Though it is now far back in point of time, I remember still having been handled by a doctor with very cold hands, and I never forgot it or forgave him. I recommend you to begin palpation with the head. You do not often palpate the head of a grown-up patient, but in the case of a child it is very important to do so. First of all go over the anterior fontanelle; notice whether it is closed or open, bulging or depressed. The fontanelle tells you as much in a baby as the pulse does in older patients, a depressed fontanelle having the same significance as a feeble pulse. Notice whether there is any sign of softening of the bone, such as one finds in craniotabes, which is a frequent sign of rickets, or syphilis. Observe also whether there is any bossing of the head, and whether there are any nodes or lumps upon the bones.

Having studied the head, you can proceed to the trunk, passing your hand over the skin, and noticing whether it is hot or cold, dry or moist, for the latter is information which no thermometer can ever give you. You will also notice whether there is beading of the ribs, which is an important and early sign of rickets; then feel for the edge of the liver. You can generally make it out quite well a little below the costal margin. The spleen, of course, you ought not to be able to feel in health. Lastly, I think you should run your hands over the limbs and notice whether there is any tenderness of the legs. Many cases of obscure disease in children are to be explained by inflammatory affections of the bones, which can only be determined by careful palpation.

We pass next to **auscultation.** One should perform this before percussion in children, reversing the rule which obtains in the case of older patients. Percussion is apt to frighten children, and so would make auscultation difficult if that were left to the last. In auscultating the chest it is best to have the child sitting up; he should not be lying on his face, for the child is likely to have unpleasant associations with that position, and also because the pressure on the abdomen, if the child is on his face, pushes up the abdominal organs and interferes with the expansion of the lungs. So examine the child when sitting up and leaning against the mother's or the nurse's shoulder. It is commonly recommended that you should listen to the chests of children without a stethoscope, by putting your ear to the back. Certainly by that means you frighten the child less than by any other method, but it is open to objection in patients who are not very clean. If you use a stethoscope it must be a binaural. You have to follow the movements of the child as he wriggles about, and you cannot do that with a single stethoscope. Further, do not have a stethoscope with a metal end-piece; use vulcanite, or something which is warm when put upon the skin. It is an advantage to have a short chest-piece, so that you can get in between the mother and the child, and get round corners without interfering with the child's position. It is also of help to have a reversible chest-piece with a narrow end to go into the small spaces between the ribs. You may think that the crying of the child is a great bar to auscultation, and y [illegible] ill find it so as a beginner; but by-and-by when you ge [illegible] actice you will rather prefer that the child should cry a little, because crying insures full expansion of the lungs, and is the equivalent in the adult to the taking of a deep breath.

You will be struck at first by the length of time that a child can hold his breath, and you may have to wait for quite a long time for another inspiration. This is not a sign of disease; it is merely due to the enormous relative vital capacity of the infant chest. There is another peculiarity of respiration met with in children who are the subjects of serious respiratory disease which is likely to confuse you at first, and which is best described as an *inversion of the respiratory rhythm.* You and I, when we breathe, first fill the chest, then empty it, and then stop for a moment;

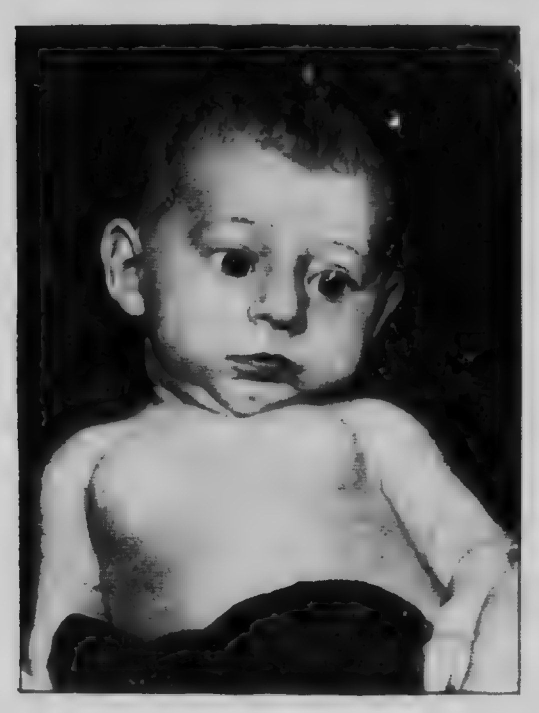

FIG. 1.

SWELLING OF THE BREASTS IN A NEWLY BORN CHILD.

that is to say, the ordinary rhythm is inspiration, expiration, pause. But you will often notice in children in whom respiration is embarrassed that a short grunting expiration is made, followed by a full inspiration, and that by a pause. In other words, the ordinary rhythm has become inverted. What the advantage of this mode of breathing is it would be hard to say. It has been suggested that the result of such breathing must be to facilitate free oxidation of the blood, for the pause after inspiration means that the lungs are kept full of pure air for a much longer period than by the normal method; but it must be confessed that this explanation is not very convincing. Such a reversal of the usual mode may be possible to the child by reason of the greater plasticity of his respiratory apparatus, and by the fact that the mechanism of respiration is not so stereotyped as it becomes in the adult. At all events, whatever the explanation may be, you will find the occurrence of this inversion of respiration of considerable diagnostic value in many cases.

There are other points in connection with auscultation of the lungs in children which have to be noticed, and one of these is the harshness of the respiratory sound. This is the so-called puerile breathing, and it is so marked and constant that, if you hear loud breathing on one side of the chest and faint on the other, the loud is probably the normal. Towards the right apex, and between the scapulæ behind, the respiration in the young child is normally not only harsh, but almost bronchial, a point which it is well to remember if one is to avoid errors of diagnosis.

Another noticeable thing is the extreme ease with which sounds are conducted in a child's chest, so that if you hear a faint accompaniment on one side it is possible that it is really being produced on the other. In the chests of children, too, you meet with pathological conditions which you do not encounter in grown-up persons; for example, collapse of the lung. It is one of the first things to think of in considering the abnormal sounds in a child's chest, whereas it is one of the last things to consider in the case of an adult.

Passing to the **auscultation of the heart,** I would remind you that the blood-pressure in the child is low. In a young infant the capacity of the heart is to the cross diameter of the arteries as 25 is to 20. At the age of puberty the proportion is as 290 is to 61. This means that there is a relatively large channel for the blood to pass into in the child, and that there is no great obstacle opposed to the heart; hence the low blood-pressure. In consequence of this the first sound is louder than the second in all the areas, for owing to the low blood-pressure the second aortic sound is comparatively feeble.

There is also a difference in the child between the relative loudness of the pulmonary and aortic second sounds at the base. In the adult the aortic second sound is accounted louder than the pulmonary. In the infant that is not the case. Below the age of four—and this is based upon the investigation of 1,000 cases*—the pulmonary second sound is decidedly louder than the aortic. From twenty to forty, careful observation shows that the sounds are about equal. Above forty the aortic second sound becomes progressively louder than the pulmonary. This means that if you are listening to the heart in a young child you are to regard as accentuation of the aortic second sound anything which is equal to the pulmonary.

You will also note that the rhythm of the heart in little children is often irregular, and you must not attach any importance to that, particularly if the child is asleep. Note also the comparative frequency of congenital bruits. If you hear a loud murmur below the age of two it is almost certainly congenital, because endocarditis hardly ever occurs at that age. Hæmic murmurs, however, may occur below two years. This has been denied by some writers, but there can be no doubt of the fact, and such murmurs may be mistaken for the results of organic disease.

The study of the **pulse** is of little importance in the case of young infants. If you want to count the rate of the heart you do it by listening with the stethoscope, and if you want to learn anything about the tension of the blood you do it by observing the fontanelle.

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* See a paper by Miss Sarah Creighton, M.D., in the *Medical Record*, N.Y., 1900, lvii., p. 45.

Lastly, we come to **percussion,** and here let me say at once that it is not necessary to use a heavy stroke. Do not percuss the chests of little children forcibly. You learn more by light percussion, and you do not give the child the impression that you are punishing him. It is often an advantage to use three fingers, for by that means you throw a broad area into vibration. I ask you to remember also that you may get certain sounds normally in infants which are abnormal in adults, such, for example, as the cracked-pot sound. You can often hear a distinct cracked-pot sound on percussing the apices of a crying child as the air is driven out from the apex of the lung through the mouth. That is due to the great elasticity of the chest-wall of the child, which yields before the percussing finger. Be careful also when you are percussing the chest of a child to percuss during the same phase of respiration when you are comparing the two sides of the chest. You will find that this makes far more difference in the case of children than it does in grown-up people, and I have known forgetfulness of this fact lead to the belief that there was dulness, whereas one was only percussing one lung during inspiration and the other during expiration. Sometimes you will detect dulness over the manubrium of the sternum in a child; and you must remember in such a case the possibility of an hypertrophied thymus or enlarged bronchial glands.

The investigation of the functions of the **nervous system** in little children is not an easy matter. *Motor paralysis* you must detect by observing whether or not the limb or group of muscles which you believe to be affected is made use of voluntarily. You cannot estimate the exact degree of paralysis by opposing passive resistance to movements as you do in the case of the adult. Remember, however, that a child may not make use of a limb from causes other than paralysis, such, for example, as pain. To this *pseudo-paralysis*, as it is called, I shall have occasion to refer in another lecture.

The *sensory functions* you must investigate as best you can on the same lines as those on which one proceeds in the case of adults, but fortunately sensory paralysis is not common in little children.

The *knee-jerks* are best tested in infants by placing the sole of the foot on the palm of your hand as on a stirrup, whilst with the other hand you tap over the patellar tendon. The *superficial reflexes* are normally rather brisk in childhood, and remember that it is normal for the sole of the foot to give an extensor response up till about the time when the child begins to walk. In other words, a Babinski reflex is normal throughout infancy.

Kernig's sign is one which is often referred to in descriptions of nervous disease in childhood. It is best elicited by placing the child on his back and keeping one leg fully extended whilst the other thigh is flexed to a right angle. If the knee on this side cannot now be extended beyond one and a half right angles (135°) the sign may be regarded as present. I should advise you however, to be very careful about making inferences from the presence or absence of this sign. Personally, I attach very little importance to it.

In examining the **fundus of the eye** in young children it is best to use the direct method, kneeling at one side of the cot with a lamp on the pillow at the opposite side of the head. No part of your investigation requires greater skill or patience than this, and you must often be content with a very fleeting glimpse of the optic disc.

The **ear** you will examine in the ordinary way, but it is well to remember the shortness of the auditory meatus in infancy and the great obliquity of the drum membrane.

Having carried out these methods of investigation, you will look lastly at e **tongue and throat.** The importance of examination of the throat of the child I cannot exaggerate, because so many diseases of children are associated with abnormal conditions of the fauces. Here you may have to use some force in order to open the child's mouth. There are many ways of coaxing a child to open his mouth, but you may not have any time to waste over him, and may have to proceed to more energetic measures. One of the best ways is to push the lower lip over the lower incisor teeth, and then to press downwards; in order to take the lip away from the teeth the child will open his mouth. Sometimes you may require to hold the nose till a gasping breath is taken. You examine the throat in the usual way.

taking care that you get plenty of light, and in many cases you may also require to proceed to palpation of the throat; but that I shall describe when speaking of the part played by adenoids in the diseases of children.

Finally, gentlemen, you must not expect to be very systematic in examining children. I have spoken as if you proceed systematically, but when you come to investigate a case you have to seize the opportunity of examining an organ when it presents itself. When the child is on his back, you must examine the heart, the anterior aspects of the lungs, and the abdomen, and when he is sitting up you have to examine the back of the lungs and the spine. You have to run over everything quickly as opportunity offers.

There are still one or two special points to be mentioned. The first is the **weight and height** of the child. I cannot impress upon you too strongly the importance of weighing babies. In the children's hospital I have every baby weighed before I see it at all. The weight is often the only criterion as to whether the infant is improving or going back in health. I do not mind what weighing apparatus you use, but you must weigh all your children so far as you can. I have put into your hands a table showing what weight a child should be at different ages, and I have here a chart showing the curve of weight for the first year (Dia. 1). There are certain turning-points of weight to which I would direct your special attention, and which I hope you will be able to remember. At birth the average weight is 7 pounds. At four months old the weight is 14 pounds, at twelve months 21 pounds, at six years 42 pounds, at fourteen years 84 pounds. These weights, being multiples, can be easily kept in mind.

Another important point is the **circumference of the head.** You should remember that the normal is 13 inches at birth; at the ninth month it is 17 inches; at one year it is 18 inches; at five years it has only grown another 2 inches. This will show you the enormous rate at which the child's head grows during the first year of life—growth which is due to the rapid enlargement of the brain.

In conclusion, there are certain **milestones,** as I may call them, in the development of the child to which I should like to draw your attention because they are points with which mothers and nurses are familiar, and which you will look foolish about if you do not know them. The first is the time or age at which the child *cuts its teeth.* This begins at the sixth month. Of course, you will look absurd if you ask whether a three-months old baby has got any teeth. All the temporary teeth are present at the age of three years. The permanent teeth begin to come at the age of six years instead of six months, and by the age of twelve all except the wisdom teeth are present. The second milestone is the *closure of the anterior fontanelle*, which occurs between the eighteenth and twenty-fourth months. If it is not closed by the twenty-fourth month there is something wrong. The third milestone is associated with the *assumption of the erect position*. A healthy baby will hold up his head at three to four months ; before this he merely waggles the head about as if trying to balance it on the neck. If the child does not hold his head up by the age I have mentioned, you should suspect some impairment of mental development. The infant sits up at from nine to twelve months and will walk at from twelve to eighteen months. He should talk well when he is two years old.

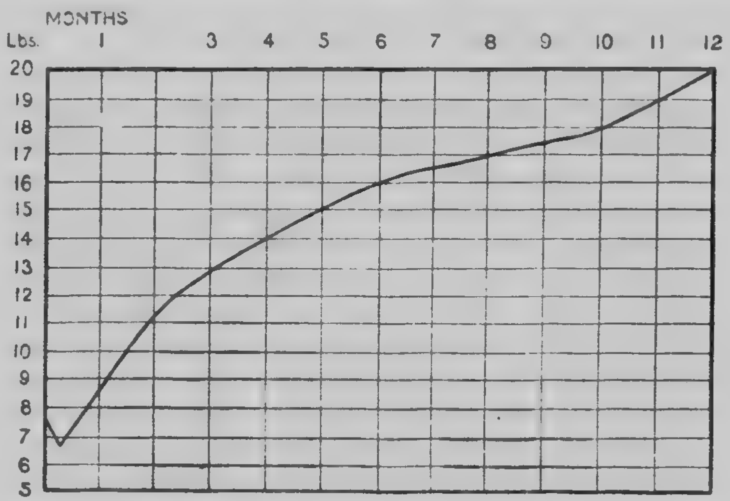

Diagram 1.—Weight Curve of First Year. (After Holt.)

The last thing I want to mention to-day is the **character**

of the motions in a child. Alterations in the fæcal evacuations constitute another set of milestones in development. Remember that for the first eight weeks there should be three to four motions daily. You are not to regard this number of motions at such an age as evidence of diarrhœa. Up to this period the motions resemble beaten-up eggs in colour and consistence, and they are of somewhat sour but not feculent odour. Look specially for cheesy masses of casein in them, for if you find these you know that the child is getting more milk than he can properly digest. Up to the end of two years from about the eighth month there is an average of two motions daily. They become now more brown in colour, more porridgy in consistence, and of a slightly feculent odour. After two years of age the motions are well formed and feculent.

In my next lecture I shall deal with some of the commoner diseases which affect the newly-born child.

LECTURE II

SOME DISEASES OF THE NEWLY-BORN

GENTLEMEN,—So soon as a child is born it meets with various adverse influences, which may render it the subject of disease before it has hardly begun to live. As many of these diseases are capable of being prevented if only one is alive to the possibility of their occurrence, I propose to devote this lecture to a brief consideration of some of the commonest of them, only premising that I shall not attempt to deal either with surgical ailments or with congenital malformations or deformities.

At the outset it may be well to recall to your recollection some of the outstanding physiological peculiarities of the newly-born child, some of which render it peculiarly vulnerable to the attacks of disease.

1. For the first few days at least of life the heat-regulating mechanism has not got into working order. In consequence of this, young infants, especially if at all premature, suffer severely from exposure to cold, and chill is a danger which must be sedulously guarded against in their case. For the same reason, rises of temperature are apt to occur upon slight provocation, and have not as much significance as they have later on. A slight rise of temperature in the first three days of life may, indeed, be considered as a normal occurrence. Should the rise be great (103° F. or so), it is to be regarded, in the absence of any other apparent cause, as an indication for the necessity of giving food. To this form of pyrexia the term **Inanition Fever** is sometimes applied.

2. The substitution of pulmonary for placental respiration is one of the most important changes incidental to the

beginning of extra-uterine life. Any hitch in the establishment of lung-breathing leads, as you know, to the appearance of **asphyxia,** the modes of overcoming which you learn when studying midwifery. You must remember, however, that even although an infant may start to breathe with its lungs, it may not use them to their fullest capacity, owing to portions of the lungs failing to expand or undergoing collapse again from feebleness of the respiratory movements. To this state of things the term **Atelectasis** is applied. Now, atelectasis may be a serious trouble, and, further, it is not easy to recognize. Its physical signs are, unfortunately, extremely vague. At the most you are only likely to find some impairment of note at the posterior parts of the lungs, with feeble air entry on auscultation, and possibly a few fine moist sounds. Sometimes there are no definite physical signs at all. You must, therefore, depend for your recognition of the condition upon symptoms. If the infant cries feebly, if it is cyanosed, either constantly or in recurring attacks, then you should suspect atelectasis. I have said that atelectasis may be serious, and it is so because it may lead to sudde[illegible]ath. I have known this happen several times, and it [illegible]d lead you to be cautious in your prognosis. The only way to treat atelectasis is to get the child to cry vigorously, and so inflate its lungs. There is no better method of doing this than by vigorous spanking, cruel though such treatment may seem.

3. I would remind you of the great destruction of red blood-corpuscles which takes place in the first few days of life. I shall deal with this process in greater detail in another lecture (p. 301), but would only remind you now that such destruction of red cells is responsible for the production of **icterus neonatorum.** This, as you know, occurs in about two-thirds of all infants, beginning about three days after birth, and lasting, as a rule, not more than a week or ten days. It is probably brought about in this way : The great destruction of red corpuscles liberates so much blood pigment that the bile is rendered unduly viscid, and stagnates in the bile capillaries, whence some of it is reabsorbed into the circulation. This, at least, is one possible explanation. Another is that the bile so formed

does reach the alimentary canal, but some of it is reabsorbed and reaches the general circulation by the still patent ductus venosus. Which of the explanations is correct does not much matter, but it should be noted that there is no bile pigment in the urine in cases of icterus neonatorum, apparently because it is deposited in the tissues, and breaks up there. This may be of diagnostic value in distinguishing icterus neonatorum from other forms of jaundice in early life.

4. The existence of the stump of the umbilical cord is one of the most important facts in the anatomy of the newly-born child, and is a constant source of danger, as will be shown immediately. Meanwhile I would ask you to remember that it should fall off within five days, and that the raw surface left should be cicatrized by about eight days later.

5. It may be worth while to remind you that **inflammation of the mammæ** is very common in children of both sexes in about the second week of life, and that sometimes it may go on to abscess formation (Fig. 1).

We may now pass on to consider some of the commoner diseases which affect the newly-born child, beginning with

INFECTIVE DISEASES.

I have already pointed out to you that the existence of the umbilical wound is a constant menace to the health of the young infant, and it is through this channel that infection usually finds access. Its simplest form is a mere local inflammation **(omphalitis)**, which causes a little redness and swelling, with slight purulent discharge, but without any general symptoms. As a sequel to this **septic thrombosis** of the umbilical vessels may occur. This is most likely to happen in the case of premature infants, in whom closure of the vessels is apt to be slow and imperfect. From septic thrombosis it is but a step to **general septicæmia,** the supervention of which may be assumed when, in addition to umbilical sepsis, there appear such symptoms as pyrexia, profound icterus, purpuric hæmorrhages, diarrhœa, and signs of inflammation of the internal organs or serous membranes. These symptoms, of course, are not necessarily all present in one case. In some even fever is absent, and in others the initial umbilical affection may be so slight as to escape detection. Icterus and hæmorrhages, when they occur, are the expression of a profound poisoning and destruction of the blood, and are very unfavourable signs. Of the internal inflammations, peritonitis and pleuro-pneumonia are the commonest.

Septicæmia in the newly-born is, as may be imagined, a very dangerous condition, and usually proves fatal in less than a week.

The infections I have hitherto described are all due to invasion by pyogenic organisms, but specific pathogenic bacteria may also find entry to the body by the umbilical wound, conspicuous amongst which is the tetanus bacillus.

Tetanus neonatorum is, fortunately, not a common disease in this country, but it crops up every now and then in the most unexpected way. It usually begins about the fifth day, and is very rarely seen after the second week. The first symptom which will probably be noticed is that the child has some difficulty in sucking, owing to stiffness of the jaw (trismus). This is soon followed by rigidity of the limbs and slight convulsions. You will distinguish these from ordinary infantile convulsions (1) by the presence of trismus, and (2) by the fact that between the convulsive attacks there is slight general rigidity, which does not completely relax, any movement of the child being apt to bring on a fresh series of spasms.

Tetanus neonatorum, I need hardly tell you, is a very fatal disease, the mortality being probably about 95 per cent., and I have never seen a case recover myself ; but occasionally cure—or recovery—results, and it is always one's duty to institute energetic treatment.

The first thing to be done is to administer antitetanic serum *at the earliest possible moment*. Ten c.c. should be injected subcutaneously so soon as the diagnosis has been made, and this may be repeated two or three days later. Meanwhile an attempt should be made to alleviate the convulsions by the administration of chloral—5 grains by the rectum, or 20 minims of the syrup by the mouth—these doses being repeated in an hour if necessary.

Such is the ordinary treatment. Lately the administration of formalin has been recommended, and at least one apparent success has been recorded from its use, though I cannot in the least tell you how it acts. One drop of it may be administered in 50 c.c. of salt solution by the bowel, or ½ drop in 10 c.c. of salt solution hypodermically. This may be repeated daily if the case goes on.

In tetanus, as in all the other umbilical infections, prevention is better than cure, and prophylaxis can only be secured by careful attention to the stump of the cord. It should therefore be enveloped from the outset in a dry sterile dressing, and if any signs of suppuration should appear after separation, local antisepsis should be vigorously carried out. It is only in this way that these very fatal diseases can be prevented.

Of infections which find access to the body by another route than the umbilicus, I would only mention **gonorrhœal septicæmia,** which may follow upon gonorrhœal conjunctivitis, and manifests itself by synovitis, just like the so-called gonorrhœal rheumatism of adults. This I have seen more than once in newly-born babies. So far as I have observed, the prognosis is not unfavourable.

THE HÆMORRHAGIC DISEASE.

Severe infections of any sort in the newly-born are apt to manifest themselves by hæmorrhages, chiefly of a purpuric character. They are in this respect directly comparable to the hæmorrhagic form of the acute specific fevers —*e.g.*, hæmorrhagic smallpox—and have usually a fatal issue. Apart from these there is a disease peculiar to newly-born infants which manifests itself chiefly by a tendency to bleeding, and which, for want of a better name, is spoken of simply as **the Hæmorrhagic Disease.** Of the true pathology of this affection we know nothing. In some cases it is undoubtedly associated with congenital syphilis, but this is by no means true of all. It is natural to suspect an infection, but although this is possible—and, indeed, probable —no one has yet succeeded in demonstrating the presence of a constant infective agent, and the other signs of infection, such as fever and umbilical sepsis, are generally conspicuous by their absence.

The disease usually first manifests itself about the second day, and very rarely starts after the twelfth. As a rule, the first thing to be noticed is hæmorrhage from the alimentary canal. You must not expect, however, to find anything like a true hæmatemesis or melæna. The bleeding is more of an oozing than an actual hæmorrhage, and reveals itself by the vomiting of small quantities of brownish grumous-looking fluid, or by the presence of similar discharges on the napkins. Bleeding may also take place from the umbilicus, and may be profuse ; and there may also be subcutaneous hæmorrhages, like those of purpura, or bleeding into the internal organs. The latter may be difficult to detect, unless so situated that they produce definite signs or symptoms. This took place in the following instances :

CASE 1.—A boy, two days old, one of twins, seemed healthy at birth, but on the second day vomited a little brownish fluid. Soon after this he became collapsed and comatose, with a bulging fontanelle, contracted pupils, and rhythmical respiration. Hæmorrhage into the meninges was diagnosed, and puncture of the anterior fontanelle resulted in the escape of a good deal of blood. After this he brightened up a little (probably from the relief of pressure), but a few hours later slight convulsions set in, followed by death.

CASE 2.—A boy, three days old, had been born at the full time by an easy forceps labour. He suffered from asphyxia after birth, and was only resuscitated by prolonged artificial respiration. He cried a great deal during the first two days, and then became rather collapsed. When I saw him he was deeply jaundiced, with a sunken fontanelle, but with no bleeding from the umbilicus or into the skin. That morning he was said to have brought up a little blood from 'the throat.' The bowels had been opened with castor oil, and on the napkin there was a small tarry motion and a good deal of meconium. In the left side of the abdomen there was a dull swelling, difficult to define and not movable, which the doctor in attendance said had not been there that morning.

Death ensued a few hours later, and although a post-mortem could not be obtained, there can be little doubt that the swelling was due to a hæmorrhage into the neighbourhood of the kidney.

Apart from the hæmorrhages, the disease may run its course without any general symptoms, and prove fatal simply by a sort of general collapse. If the bleeding is entirely internal, or only from the alimentary canal, it may easily be overlooked, in which case the child may be believed to have died from 'convulsions,' or simply from 'prematurity.' There can be no doubt, indeed, that many cases really do pass unrecognized in this way, and that the disease causes more deaths than is properly realized.

The **prognosis** in the hæmorrhagic disease is always grave, and if the symptoms last for more than a day, is very bad indeed, although recovery *may* take place even in what seem the worst cases.

As regards **treatment** there is little to be said, for one is almost helpless. 'Styptic' remedies given internally seem to be of very little use, and although it is important to maintain the child's strength if one can, attempts at feeding are apt to induce vomiting and fresh hæmorrhages from the stomach and bowels. The administration of weak solutions of gelatine by the mouth have been said to be of use in some cases, and are always worth trying. Bleeding from the umbilicus must be controlled by pressure in the usual way, and in hæmorrhage from the skin or external mucous membranes the application of adrenalin solution is indicated. When all is said and done, however, you can do but little to influence the course of this mysterious disease, and to 'treat symptoms and trust to luck' pretty well sums up one's therapeutic resources with regard to it.*

PEMPHIGUS NEONATORUM.

It has been well remarked that pemphigus is not a disease, but a lesion, and in the newly-born child we can distinguish two varieties of it: (1) syphilitic, (2) non-syphilitic.

* Successful results have recently been reported from the subcutaneous injection of from 2 to 5 c.c. of normal human blood or blood-serum in this disease.

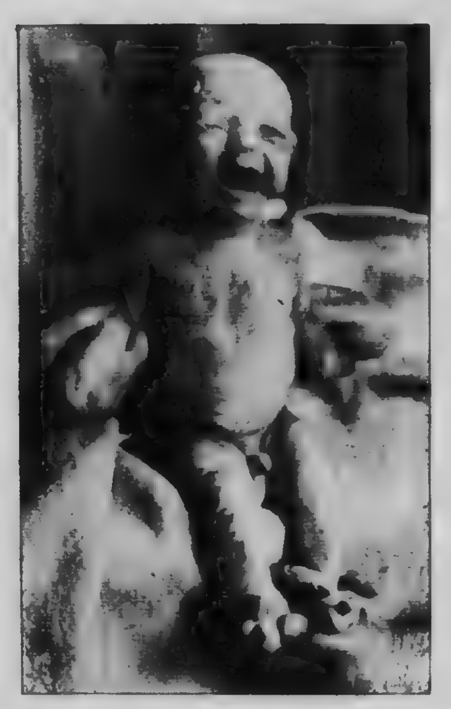

FIG. 2.

GENERAL EXFOLIATIVE DERMATITIS.

Syphilitic pemphigus is distinguished by the fact that the bullæ occur on the palms and soles, as well as on the trunk, whereas in non-syphilitic pemphigus the palms and soles escape.

Of the cause of **non-syphilitic pemphigus** we are ignorant, although it also is probably an 'infective' disease, due to pyogenic organisms, and closely allied to impetigo. In some cases at least it can be shown to be simply a cutaneous manifestation of general sepsis. Thus, of the cases which have occurred at the London Hospital within the last ten years, three were associated with umbilical infection and two with sepsis consequent upon ritual circumcision. In institutions it may occur in the form of epidemics, which is another indication of its infective character.

Apart from the eruption, which may come out all at once or in successive crops, the general symptoms vary greatly. Sometimes there are practically none at all; at others there is high fever, prostration, and symptoms of general septicæmia.

The prognosis varies in accordance with the general symptoms, but is always rather grave. Of seventeen cases admitted to the London Hospital—excluding those due to syphilis—thirteen died. In syphilitic cases the mortality is probably even higher.

Treatment in the syphilitic cases is simple, and consists in pushing mercury. In non-syphilitic cases all that one can do is to maintain the strength of the infant, and use antiseptic dusting powders, or weak germicidal lotions locally.

Somewhat resembling pemphigus is the condition known as **Exfoliative Dermatitis**—a rare disease sometimes met with in newly-born infants. The epidermis here peels off in great sheets, leaving the true skin denuded just as if it had been scalded, and producing a picture which, once seen, can never be forgotten (Fig. 2). In spite of the extensive skin lesion, there may be few or no general symptoms, but sometimes there is high fever and prostration. I cannot tell you what the mortality rate is, as the disease is a rare one, but in the few cases I have seen death and recovery took place in about equal numbers. The treatment is the same as that of non-syphilitic pemphigus.

LECTURE III

THE ARTIFICIAL FEEDING OF INFANTS

GENTLEMEN,—We approach to-day a subject which exceeds, perhaps, in importance any to which I shall have occasion to direct your attention throughout this course of lectures—I mean the subject of infant feeding. When you go into practice, the question, How is one to feed this baby ? is one which will occur to you with perplexing frequency ; and although it is a subject upon which a great deal has been written, I think one may say this, that the more you read about it the more confused you are likely to get, for, it must be confessed, much that has been written on the subject serves only to darken counsel, and you will find it of very little use to you in practice. I shall not deal in this lecture with all the conceivable methods by which children might be or have been fed ; I want rather to describe quite plainly the methods which I have tried myself, and which I think you will find most easy and simple to carry out in your ordinary work.

Now, the first question you have to ask yourself is, Why should artificial feeding of children be necessary at all ? You know that in the natural state of things every woman is the source of nourishment for her own child, but you will not have been long in practice before you realize that under modern conditions it is the minority of women who can nurse, or do nurse, their own children. It would be unjust to the female sex generally to state that this inability to nurse their children is their own fault. I do not think that is true ; I think, in the majority of cases, one can say that the woman is unable—not unwilling—to nurse her own child. Now, that is a phenomenon of very considerable gravity, but it is one which is not confined to this country; it has been found to hold good of the whole of Europe, and in America also.* It seems to be part of the price which we pay for civilization. Be that as it may, you will see from what I have said how important it is that you should understand how best to make good the absence of the mother's milk, and how it becomes a matter of pressing importance in practice.

If a woman is unable to nurse her own child, obviously the next best substitute is to get another woman to nurse it for her; in other words, a wet nurse is the best substitute for the mother. But I do not think you will have occasion to employ a wet nurse often in practice. There are many practical disadvantages entailed in that course, and, as a matter of fact, *artificial* feeding is synonymous in most cases with *bottle* feeding, so that what we have to consider to-day is how you can best feed a child by the bottle. There is still another question which has to be disposed of before we come to the consideration of the best way to carry out bottle feeding, and that is, Supposing a woman starts to nurse her own child, how are you to know whether the process is successful or not? In other words, what is to justify you in adopting artificial feeding in the case of a woman who has *begun* to nurse her own infant? Gentlemen, I know only one condition on the part of the child that justifies premature weaning, and that is *persistent loss of weight*. If a child is persistently losing weight when fed on the mother's breast, that is an indication, and an imperative one, for weaning, and there is no other imperative indication that I know of. Such a state of things is illustrated in the chart (Dia. 2). It is that of a child seven weeks old which was being fed on the breast, and as we weighed it from week to week we found it increased in weight very slowly, or hardly at all. And as there was no digestive disturbance we concluded that the mother's milk was insufficient; and we accordingly weaned the child, with the result you see—namely, that the weight shot up enormously, and the child gained more in one week of bottle feeding than it had gained in two or three weeks of breast feeding. There is one caution I should give you here. Before you conclude that the breast milk is insufficient, make quite certain that you have corrected any digestive disturbance in the child; and you will find particularly that you have to correct, in breast-fed babies, constipation. This, as I shall have to point out later on, is the vice of breast-fed babies, as diarrhœa is the vice of bottle-fed babies. So you see the statement I made that progressive loss of weight on the part of the child is the only indication which justifies premature weaning has to be qualified by this, that it only holds good in the absence of digestive disturbance.

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* See Bunge, 'Die Zunehmende Unfähigkeit der Frauen ihre Kinder zu Stillen.'

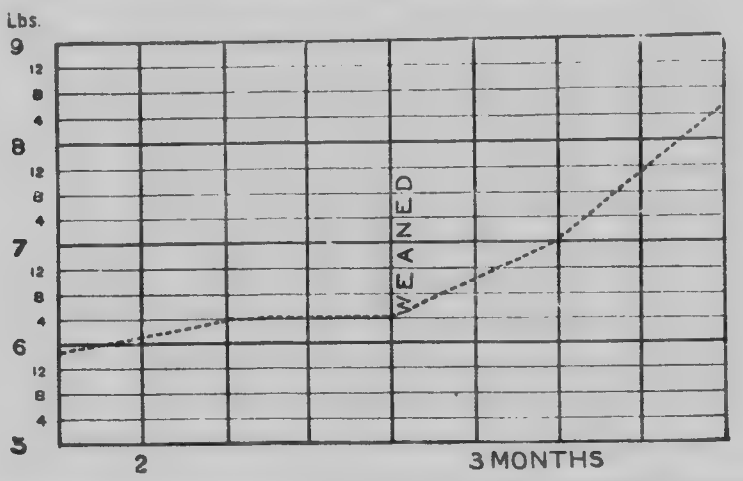

Diagram 2.—Insufficiency of Breast Milk, showing Effect of Weaning.

Having disposed of these preliminaries, we have next to consider **what substitute we can use for human milk.** Now, in practice, cow's milk will always be your substitute. I shall not consider in these lectures the merits of other forms of milk. Many other kinds have been recommended, but the practical conveniences of cow's milk are so great that one rarely uses any other. I cannot too strongly impress upon you, gentlemen the fact *that it is the casein which is the source of difficulty in the artificial feeding of children with cow's milk.* There are chemical reasons, which I shall not go into now, why the casein of cow's milk is less digestible than the casein of human mi'~ but whatever those reasons may be, it is a fact which you will meet in practice that you have difficulty in getting infants to digest cow's casein. For that reason you must give the cow's milk in a diluted form. How much, then, should you dilute the milk? It is best to begin first of all, as a simple practical rule, with a **dilution of half and half**; that is to say, give as much cow's milk as water. If you like, you can use barley-water or lime-water as the diluent; these make it more easily digestible than plain water. If you compare a mixture so prepared with human milk, you find that from a chemical point of view it works out as follows: In human milk there is 1½ per cent. of protein, 6½ per cent. of sugar, and 3½ per cent. of fat. In cow's milk there is 3 per cent. of protein, 4½ per cent. of sugar, and 4 per cent. of fat. If you dilute with equal parts of water you have 1½ per cent. of protein, 2¼ per cent. of sugar, and 2 per cent. of fat. This makes the proportion of protein, roughly speaking, correct, but the sugar and the fat are too poor, and you must therefore make up the deficiency of these constituents. That you can do quite simply by adding two teaspoonfuls of cream and two level teaspoonfuls of sugar to every 6 ounces of the mixture; that is assuming the cream to contain the average of fat of London centrifugal cream, which is about 45 per cent. That gives you a mixture which quantitatively is a very fair approximation to mother's milk. Qualitatively, of course, it is not the same thing as human milk, and it never can be.

The next question is, **How much** of the mixture shall you give, and **how often?** I have put into your hands a table showing you the quantities which the child's stomach will accommodate and the proper intervals of feeding. You must not suppose that the quantities there given should be quite hard and fast; you must leave it to some extent to the baby's appetite. It is usually safe to tell the mother to give the child as much as he will take at each feed.

The next point is, What sort of **bottle** should you use? The only sort of bottle to be recommended is a boat-shaped one with a rubber teat attached.* The rubber tube bottles so much used probably destroy more infant life than any other single agency, and it would be a good thing if their use were illegal here, just as it is in France and in many States of America. The screw-top bottles are not much better. Impress upon the mother that the bottle must be kept very clean. With regard to the teat, many cases of failure are due to its having too small an aperture in the top. You may want to know when the aperture is big enough. It is big enough if, when you hold the bottle upside down, the milk drops slowly, at the rate of about 1 drop per second. It ought not to require a great deal of sucking on the part of the baby, or it will be apt to give the child flatulence, or the infant will leave off sucking before he is satisfied, and so cease to gain weight.

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* The Oval Hygienic Feeder and Maw's Universal Feeding Bottle are both good patterns.

TIME-TABLE FOR INFANT FEEDING.

Age.	Intervals by Day.	Night Feeds.	Quantity at each Feed.
	Hours.		Ozs.
1 week	2	2	1 to 1½
2 to 3 weeks	2	2	1½ to 3
4 to 5 ,, ...	2	1	2½ to 3½
6 to 12 ,, ...	2½	1	3 to 4½
3 to 5 months	3	1	4 to 5½
5 to 9 ,, ...	3	—	5½ to 7
9 to 12 ,, ...	3½	—	7½ to 9

The next point is, **Should the milk be boiled,** or should it be given raw? That, gentlemen, is a point which has probably been more discussed than its importance really justifies. I advise you to give it neither boiled nor raw, but scalded. That may appear to you to be a distinction without a difference, but by scalding I mean raising the milk to the boiling-point, or just short of that, and then taking it off the fire. The drawback to boiling the milk is, that there is a very slight risk of producing scurvy by it. But that can be easily remedied by adding fruit juice to the diet. In hot weather it is much more necessary to scald the milk, because thereby one diminishes the tendency to its going sour and causing diarrhœa.

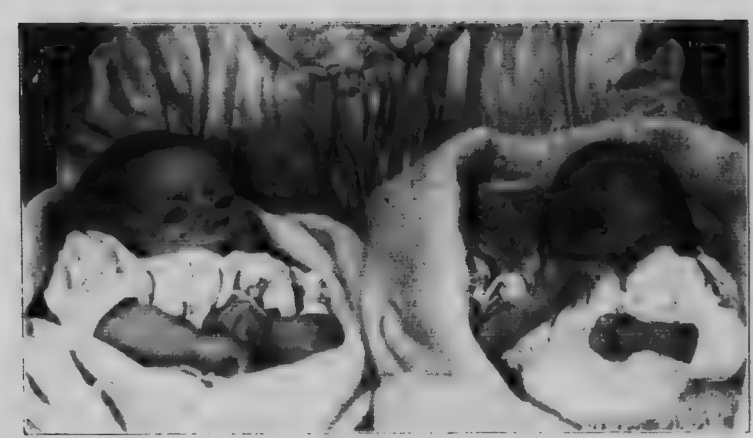

FIG. 3.

TWINS, THE ONE ON THE LEFT BREAST-FED; THE OTHER BOTTLE-FED.

A commentary on the advantages of breast-feeding.

Having given the food by the bottle I have described, and at the intervals and in the proportions pointed out, you have to ask yourself, **How are you to know whether the results are satisfactory?** Here, again, the only test that the mixture is suitable is the balance. At what rate, then, should the child gain weight? This is not difficult to remember, because a healthy child should gain almost 1 ounce a day; he should gain 6 ounces a week. Obviously, one of two things will happen on this mixture: either the child goes on gaining weight, or he suffers from digestive disturbance, colic, possibly diarrhœa, possibly vomiting. If the child does not gain weight, or if he suffers from digestive disturbance, what are you to do? I advise you to remember the possibility, particularly if you are dealing with a very young child, that this **mixture is too strong,** and that it may be advisable to make it more dilute. In this chart (Dia. 3) I show you an instance of such a result. It is the chart of a child eleven weeks old who was fed on milk and water, equal parts of each, which resulted in the production of diarrhœa and vomiting, and only a slight increase in weight. Then we gave 1 part of milk and 2 parts of water, and immediately the weight shot up and everything went well. This shows that it is possible for a child to lose weight because it is getting too much to eat. Very often you will find that in young infants you may require to begin with 1 part of milk to 2 parts of water instead of equal parts of each, sugar and cream being added in proportion.

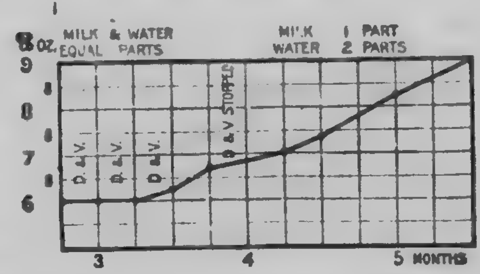

DIAGRAM 3. —FAILURE TO GAIN WEIGHT DUE TO FEEDING WITH TOO STRONG A MIXTURE.

Supposing that even in this very dilute form **the milk still disagrees,** what are you to do then? I do not think you are likely to effect much good by diluting cow's milk to a greater degree than this. ? One does not often see children do well on a mixture which is weaker than 1 in 3.

Therefore, what you have to do is to look out for a more digestible form of milk than ordinary cow's milk. You will find that more digestible form in **condensed milk.** If, then, you fail to improve the child's weight by giving it a food consisting of 1 part of cow's milk to 2 of water, I recommend you to try condensed milk. I think some consider it a heresy to recommend the feeding of infants with condensed milk at all. That is not a just position to take up. Condensed milk is an extremely useful *temporary expedient* ; if used with suitable precautions, particularly if the deficiency of fat be rectified, it may be quite successfully given for the first two or three, or even more, months of the child's life. It is quite true that the continued use of condensed milk is almost certain to result in the production of rickets if kept up until the child is six months old. But, fortunately, before six months the child's digestion has generally become capable of dealing with ordinary cow's milk.

Well, what brand of condensed milk should you use ? Never select skimmed or separated condensed milk at all. The brand selected should contain all the original cream. There are two classes of such condensed milk, those which are sweetened and those which are unsweetened. I used to recommend unsweetened, because in theory that is the best form, but I have given it up because it is so difficult to obtain. In practice I always recommend Nestlé's milk ; it contains 13 per cent. of fat—that is to say, all the original fat of the milk—besides considerable quantities of added sugar. The dilution I recommend to start with is one teaspoonful of condensed milk to six tablespoonfuls of water, not barley-water, but ordinary water which has been boiled and then allowed to cool. Such a mixture has the following composition :

Protein	1·13	per cent.
Fat	1·28	,,
Sugar	6·72	,,
Ash	0·25	,,

The total sugar is somewhat in excess, and the fat is distinctly too low ; but by the addition of a teaspoonful of centrifugal cream the total fat is brought up to 3·1 per cent., which is nearly the same as that in human milk. I have here charts which show how successful feeding may be with condensed milk. Here is one of a child which was given two teaspoonfuls of condensed milk to eight tablespoonfuls of water, and you see how steeply the weight curve rises (Dia. 4).

You may ask, Why is condensed milk so much more easily digested than ordinary cow's milk? I think it is largely because one gives it so dilute. The dilution of 1 in 24 means that the casein will be much diminished, and if you add acetic acid to such a mixture and compare it with peptonized milk, you find you get about an equal precipitate in each case.

Instead of using condensed milk you might try **citrated milk**, which is prepared by adding 1 grain of citrate of soda to each ounce of cow's milk, and give this diluted in the same way as ordinary cow's milk. The citrate of soda removes the excess of lime-salts from the milk, and prevents its forming a dense curd. You will find that milk so prepared is sometimes digested when ordinary diluted milk is not.

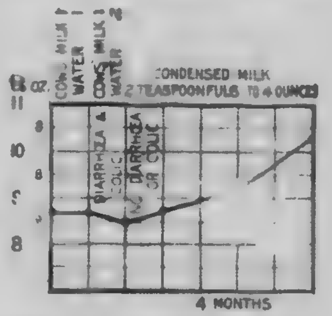

DIAGRAM 4. SUCCESSFUL FEEDING WITH CONDENSED MILK.

Desiccated milk is certainly much more digestible than ordinary milk, for in the process of drying the casein seems to undergo some physical alteration, which prevents its subsequently forming a dense clot. The best form of it I know is the preparation known as *Glaxo* (see Table, p. 37) which is often well digested even by delicate babies.

If all these methods fail, I should advise you to give fully **peptonized milk.** The best way of preparing it is by means of Fairchild's Peptogenic Milk Powder. Do not follow the directions upon the bottle, but give 8 of milk to 12 of water to begin with, and peptonize for twenty-five minutes, or longer if necessary. Nor should cream be added at first, although you are told to do so on the label. By varying the proportions of milk and water and the length of the peptonizing, you have it in your power by means of this preparation to prepare a milk of almost any degree of digestibility, and you will certainly find it of great help.

There are many charts on the wall which show the results of successful feeding with peptonized milk. This, for instance (Dia. 5), was the case of a child which had colic and constipation when fed on ordinary milk. Then we put him on to peptonized milk and water, and you see the weight rose very rapidly. Some people raise an objection to peptonized milk ; they say you will so demoralize the child's stomach by that means that it will be unable to digest ordinary milk later on. I believe, gentlemen, that this is a purely imaginary objection. I have fed children upon peptonized milk for many months, but I have never had any difficulty in getting them to digest ordinary cow's milk later on. So I think you may dismiss this bogey from your minds.

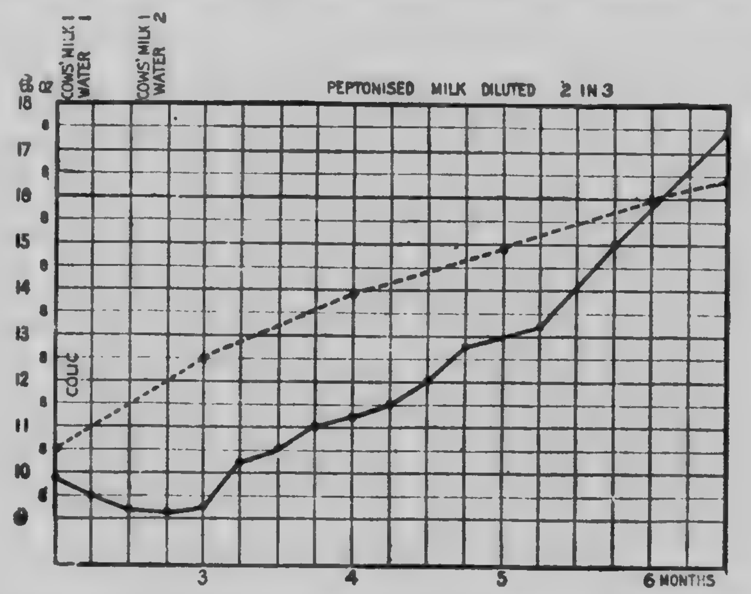

DIAGRAM 5.—SUCCESSFUL FEEDING WITH PEPTONIZED MILK. THE DOTTED LINE REPRESENTS THE NORMAL WEIGHT.

But **if you fail even with peptonized milk,** what then ? Well, gentlemen, the resources of civilization are not yet exhausted ; there are still some things you can do, and I should recommend you in a case like that, when you have tried peptonized milk and the child still does not gain weight, to try the effect of a little gray powder. I do not know how the gray powder acts in those cases, but I am satisfied that in many cases, even although there is no reason to suppose there is a congenital syphilitic taint, the administration of gray powder will in some mysterious way make all the difference to the child's digestive power, and I have charts which illustrate that fact. Gray powder seems to fatten some children, just as cod-liver oil does. This child was fed upon milk and barley-water in equal parts, but he was not doing well. Gray powder was then given, and you see how great improvement resulted, although in that case also a history of syphilis was extremely doubtful (Dia. 6).

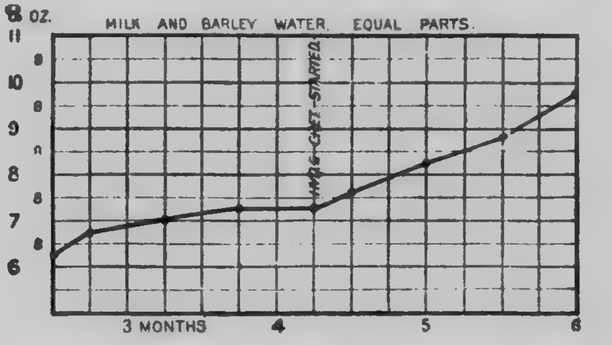

Diagram 6.—Showing the Effect of Gray Powder.

If in spite of all you do the digestion does not improve, you are justified in thinking you have to deal with a child who is incapable of digesting cow's casein. There are such children, those who cannot digest the casein, no matter how you present it to them. If you cannot get a wet nurse, in such a case you must take away the casein altogether, and to do that the simplest way is to prepare **whey** from the milk. Curdle the milk with rennet, and allow it to set into a solid mass; then stir it up and strain it through muslin.* By that means you have freed the milk from the casein. Whey is insufficient nourishment alone, because it contains very little fat, so you have to supplement the fat. The best way to do that is to add 1 part of cream to 6 or 8 of whey. Instead of using cream to supplement the deficiencies of whey you may use egg-white in some of the foods or one of the patent foods. I have used Mellin's food and whey successfully. Recently I had a case under my care in which I ultimately succeeded with Savory and Moore's food made up with whey when everything else failed ; but usually whey and cream will answer best. By such means as this you will in most cases be able to tide the child over his trouble, even if he be of very feeble digestive capacity, until such time as he has cut some teeth. When the child reaches the first dentition often a complete change comes over the digestion, and instantly his nutrition improves. It would seem as if some of the digestive glands only attain their full functional power when the first teeth are cut.

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* The whey should be scalded before use, to destroy the rennet which it contains.

You must not be surprised nor blame yourselves, gentlemen, if you find every now and then that there are cases in which you cannot find anything on which the child will thrive. There will be a residuum of cases of this kind, which will be smaller the more skilled you are in your feeding. But there *is* a residuum with whom nothing succeeds. These are cases which I shall have to speak of when I come to talk about **marasmus** ; they go progressively downhill, and they die without one being able to find out what was the cause of the fatal result. Here I show you a chart (Dia. 7) which illustrates the course of such a case, in which we tried milk and water in equal parts ; we tried it stronger and it produced diarrhœa ; we tried peptonized milk for a time, and it was succeeded by diarrhœa and vomiting, and then the child died. Nothing which you can find after death will account for this congenital feebleness of digestive power.

I now want to impress upon you one further point about the artificial feeding of babies, and that is *that you should not lightly change from one kind of food to another.* You will constantly be having children brought to you whose mothers say they have tried everything. In such a case you may generally take it that nothing has really been tried in the proper sense of the term. You cannot judge from one week's experience what any particular food is going to do for a child ; you have to keep the child on a diet for a reasonable time, so as to give it a fair trial, and it is only if you find that he is not gaining weight that you are justified in concluding that the food he is having is not suitable for him.

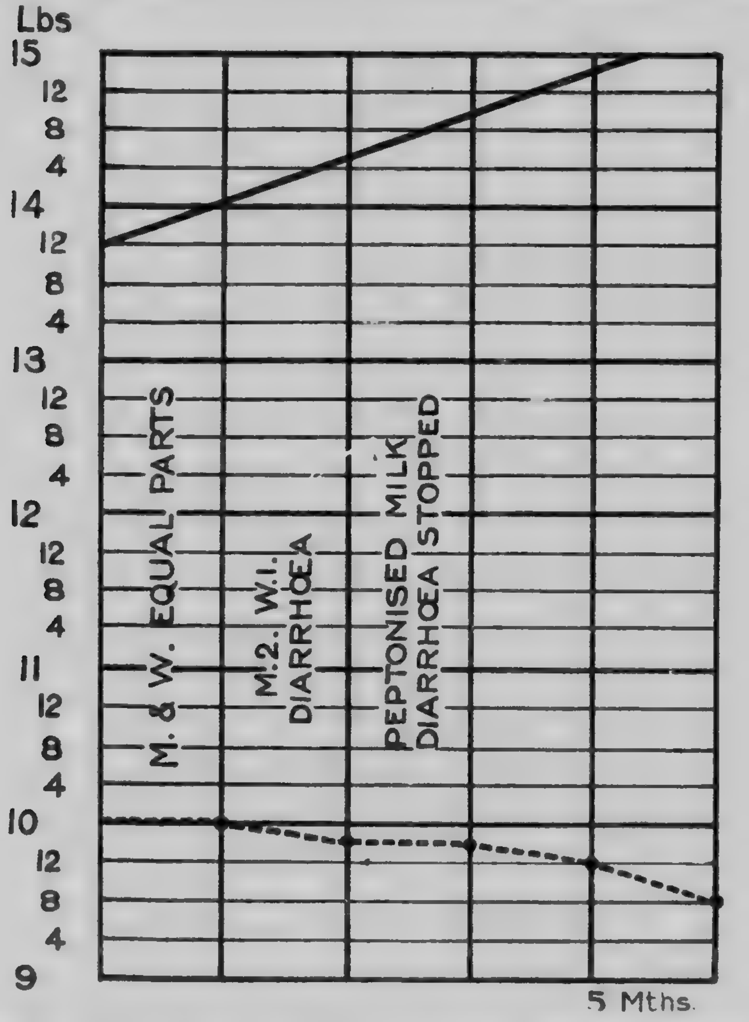

DIAGRAM 7.—WEIGHT CHART OF A CASE OF MARASMUS ENDING FATALLY. THE UPPER LINE REPRESENTS THE WEIGHT OF A HEALTHY CHILD OF THE SAME AGE.

Another question is, What should be your attitude towards the **patent foods** which are so much in use for infant feeding ? I have put into your hands a sheet which gives the composition of those foods, from which you will see that

TABLE SHOWING THE COMPOSITION OF INFANT FOODS.

Food.	Water.	Protein.	Fat.	Carbo-hydrate.	Minera Matter.	General Description and Remarks.
	Per Cent.	Per Cent.	Per Cent.	Per Cent.	Per Cent.	
Dried human milk . .	—	12·2	26·4	52·4	2·1	The standard of composition to which artificial substances should conform.
	GROUP I.					
Allenbury No. 1 (for children below the age of three months)	5·7	9·7	14·0	66·85	3·75	Desiccated cow's milk from which the excess of casein has been removed, and a certain proportion of soluble vegetable albumin, milk, sugar, and cream added. No starch present. Half an ounce in 3 ounces of water for a child aged three months.
Allenbury No. 2 (for children of the age of from three to six months)	3·9	9·2	12·3	72·1	3·50	Resembles the above, but contains some malted flour in addition. No starch present. One ounce in 6 ounces of water for a child aged six months.
Horlick's Malted Milk	3·7	13·8	9·0	70·8	2·70	A mixture of desiccated milk (50 per cent.), wheat flour (26¼ per cent.), barley malt (23 per cent.), and bicarbonate of soda (¾ per cent.). Contains no unaltered starch when mixed. Three teaspoonfuls (equals 22 grammes) in 4 ounces of water for a child aged three months.
Carnrick's Soluble Food	5·3	13·6	2·5	76·2	2·20	A mixture of desiccated milk (37½ per cent.), malted wheat flour (37½ per cent.), and milk sugar (25 per cent.). When prepared according to directions, the casein is partially digested, but a considerable amount of unchanged starch is left. One part to be mixed with 9 parts of water, and boiled for a few minutes.

Muffler's Food ..	4·7	15·1	5·1	72·1	3·00	Prepared from milk, eggs, Aleuronat, lactose, butter, sugar, and wheat flour. Contains no unaltered starch.
Glaxo	3·5	22·2	27·4	41·0	5·90	Prepared from dried mik with the addition of cream fat and lactose. All the carbohydrate in the form of milk sugar.
Milo Food	3·6	14·0	5·2	75·3	1·90	A mixture of desiccated Swiss milk, baked wheat flour, and cane-sugar (30 per cent.). Contains about 15 per cent. of starch.
Manhu Infant Food..	8·8	8·7	5·6	75·9	1·00	A mixture of desiccated milk and malted cereals. When prepared according to directions contains a good deal of unaltered starch. A dessertspoonful (equals 13 grammes) to be mixed with 2½ ounces of water.
	Group II.—*Class A.*					
Mellin's Food ..	6·3	7·9	Trace	82·0	3·80	A completely malted food. All the carbohydrate in a soluble form. May be regarded as a desiccated malt extract. Half a tablespoonful (about 5 grammes), ¼ pint of milk, and ¼ pint of water for a child under the age of three months.
Cheltine Maltose Food	4·6	5·3	0·27	87·6	2·25	A fully malted food, containing no starch. To be used with milk.
Hovis Babies' Food No. 1	3·7	7·7	0·20	86·6	1·82	A fully malted food. Carbohydrates entirely in the form of maltose and dextrins. To be made with milk.
	Class B.					
Savory and Moore's Food	4·5	10·3	1·4	83·2	0·60	Composed of wheat flour with the addition of malt. When prepared according to directions, most, but not all, of the starch is converted into soluble forms (chiefly dextrins). One or two tablespoonfuls (equals from 1 ounce to 2 ounces) to be mixed with two or three tablespoonfuls of cold milk or milk and water, and ½ pint of boiling milk, or milk and water, to be added.

TABLE SHOWING THE COMPOSITION OF INFANT FOODS—*Continued.*

Food.	Water.	Protein.	Fat.	Carbohydrate.	Mineral Matter.	General Description and Remarks.
	Per Cent.	Per Cent.	Per Cent.	Per Cent.	Per Cent.	
Benger's Food ..	8·3	10·2	1·2	79·5	0·80	A mixture of wheat flour and pancreatic extract. When prepared according to directions, most, but not all, of the starch is converted into soluble forms. The protein is also partially digested as well as that of the milk used in mixing it. One tablespoonful (about 1 ounce), and four tablespoonfuls of cold milk, then add ½ pint of boiling milk and water; set aside in a warm place for fifteen minutes, then bring to the boil.
Theinhardt's Soluble Infants' Food	5·0	16·0	5·0	70·5	3·50	Prepared from cow's milk, malt, various cereals, and sugars. Contains very little unaltered starch.
Alienbury Malted Food	6·5	9·2	1·0	82·8	0·50	A mixture of wheat flour and malt. When prepared according to the directions it still contains some unaltered starch. Designed for children above the age of six months. One tablespoonful (about 1 ounce), a teaspoonful of sugar, and three tablespoonfuls of cold water; mix and add ½ pint of boiling milk and water (equal parts).
Diastased Farina ..	8·3	7·6	1·3	81·7	1·10	A malted farinaceous food. When prepared according to the directions, practically all the starch is converted into soluble forms. One ounce of food, ½ pint of cold milk, and 2 ounces of water. Heat slowly till it boils; boil three minutes, and sweeten if desired.

Coombs's Malted Food	7·9	12·1	2·8	76·8	0·40	A malted farinaceous food. When prepared according to the directions it still contains much unaltered starch.
Moseley's Food ..	10·8	11·0	0·92	76·4	0·94	Complete conversion of all starch occurs during mixing. To be given with milk.
	GROUP III.					
Ridge's Food.. ..	7·9	9·2	1·0	81·2	0·70	A baked flour, containing only 3 per cent. of *soluble* carbohydrates, the remainder being starch. Recommended to be made with milk or water. Made with water alone is totally insufficient food.
Neave's Food ..	6·5	10·5	1·0	80·4	1·60	Resembles the above, but recommended to be made with milk and water.
Frame Food Diet ..	5·0	13·4	1·2	79·4	1·00	A thoroughly baked flour to which have been added cane-sugar and some extract of bran. It is *not* specially rich in mineral ingredients, but nitrogenous matters are abundant, and it contains much unaltered starch. One-third of an ounce to be mixed with a breakfastcupful of milk and water (1 part of milk to 2 parts of water).
Bananina	9·5	4·1	0·40	84·0	2·07	A highly starchy food prepared from banana flour.
Cheltine Infants' Food	7·2	16·2	3·92	71·0	1·83	Contains starch when prepared for use.
Hovis Food, No. 2 ..	2·4	5·7	0·10	90·1	1·70	Contains about 7½ per cent. of starch. To be made up with milk.
Robinson's Groats ..	10·4	11·3	1·6	75·0	1·70	Ground oats from which the husk has been removed. Rich in protein and mineral matter.
Robinson's Patent Barley	10·1	5·1	0·9	82·0	1·90	Ground pearl barley, poor in every element except starch and mineral matter.
Chapman's Whole Flour	8·4	9·4	2·0	79·3	0·90	A finely ground whole-wheat flour. Not much superior in nutritive value to ordinary 'households' flour. Starch entirely unaltered.
Scott's Oat Flour ..	5·8	9·7	5·0	78·2	1·30	A fine oat flour. Somewhat inferior in nutritive value to 'groats.' Starch unaltered.

many of them are entirely unsuited to be a complete substitute for human milk. I have divided them, for convenience, into three groups: First, one which is intended to be a complete substitute for human milk; secondly, a group which is intended to be made up with cow's milk, and in which the artificial part of the food is predigested; the third class includes those which contain unaltered starch. My advice to you would be to rule out the third group at once for all children under six months old. As regards the first group, they really present very little advantage over condensed or peptonized milk, and I hardly ever have recourse to them. I admit that some children may be reared successfully on certain patent infant foods, but hardly ever if condensed milk and other methods of feeding have been properly tried and have failed. They all incur the risk of producing scurvy. Therefore, if you use those foods it is well to see that some fresh elements are introduced into the diet, such as grape or orange juice.

The second group of foods is sometimes useful for children who are getting towards the period of first dentition, and who are not thriving very well because it is difficult to get them to take sufficient cow's milk. In these cases it is advisable to supplement the milk with some other food, and a dextrinized food is best. There is one class of those dextrinized foods in which the change in the starch takes place during mixing. I think it is more convenient to use a food in which the change has already taken place; and so, if one has occasion to use this group at all, one selects Mellin's food or Hovis food as being already dextrinized in preference to the others. But remember they are all deficient in fat, and their continued use is apt to result in rickets. If you use them at all it should be with that danger clearly before your minds, and they should be given only for a limited time.

One or two other questions I must discuss briefly. If a child has been on the breast, and you have not had recourse to any artificial method, and everything is going on well, **when are you to wean?** I want you to have clear ideas on this matter. It used to be said that a child should not be weaned until he has cut some of his teeth. I think even

Trousseau went so far as that ; he was guided in his weaning of children by the dentition. But you must remember always that a child may fail to cut his teeth because he is rickety, from being fed on the breast too long ; therefore, you see, the appearance of the teeth alone is no sufficient criterion of when you are to wean. You may lay it down as a general rule that the child should be weaned between the tenth and the twelfth month. Mothers, especially in the lower classes, often keep on suckling their children, partly from motives of economy and partly to prevent another pregnancy, for too long a time. I have even known children fed mainly on the breast until they were two years old, and without obvious harm. But I think in such a case there is very likely to be detriment to the *mother's* health. Between the tenth and twelfth month is the rule which you should adopt for yourselves.

Another piece of advice which I should like to give you in this matter is, never to wean a child in hot weather if you can avoid it. The reason for this is that children who are weaned in hot weather are very apt to become the subjects of epidemic diarrhœa, and to be carried off by it. It is better to allow a child to be nursed a little too long than to wean him at a time when diarrhœa is prevalent.

A kindred question to weaning is, When should you add **starch** to the diet ? When is it safe to begin to allow a child to have other food than milk ? Experience shows one that the power of digesting starch varies enormously in different cases. I have known children who got starchy food from their earliest days, and yet, apparently, did not suffer from it. I recall, for instance, the case of twins being brought to me who had been fed with a strong decoction of barley-water *without any milk* for three months, and yet they were not so thin as you would suppose, and had no digestive disturbance. I have also known a child fed upon Robb's biscuits, which contain a good deal of starch, without any harm, when one month old. On the other hand, one sees children in whom starch appears to produce fermentation, acidity, diarrhœa, and colic. So your general rule should be not to allow a starchy food in any shape until the child has cut a tooth or two, and then you can begin to give such foods very gradually and cautiously. There are some children, however, in whom it may be worth while to run the risk of giving a little starch sooner than that. Children who are not getting on well with milk, and who have a difficulty in digesting casein, should have the benefit of the trial of a little starchy food. I once got a lesson in this matter from a mother whose child I was attending for difficult digestion. We were struggling on with peptonized milk, and making no headway. One day, a week after I had seen the child, he was brought back weighing a pound more than at the last visit, and it transpired that the mother had, of her own accord, added a considerable quantity of cornflour to the diet, and with complete success. The starch-digesting capacity of that child was evidently in excess of his milk-digesting capacity, and in this case the experiment was justified by the result. Anyhow, it is an experiment which cannot do much harm, because so soon as you find it disagree you can stop the food.

I may conclude this lecture by directing your attention to some standard dietaries suitable for children at and after the period of weaning :

DIET FROM NINE TO TWELVE MONTHS.

FIRST MEAL (7.30 a.m.) : Milk thickened with groats, oat-flour, Chapman's whole-wheat flour, Mellin's Food, Savory and Moore's Food, or Allenbury No. 3.

SECOND MEAL (10.30 to 11 a.m.) : Warm milk, pure or diluted with one-third of lime-water.

THIRD MEAL (1.30 to 2 p.m.) : Warm milk, with the addition of the yolk of a lightly boiled or raw egg. A little veal broth or good beef-tea occasionally as a change.

FOURTH MEAL (5 p.m.) : Same as first.

FIFTH MEAL (9 to 10 p.m.) : Warm milk.

During this period the use of a bottle should be gradually discontinued, and the child fed with a spoon or accustomed to drink out of a feeding-cup or ordinary cup.

The amount of milk taken in the course of the day should not exceed 2 pints. If the child be thirsty, he may be given a little water between meals.

From about the tenth month onwards something to chew should be given occasionally, such as a rusk or crust, or a piece of sponge-cake, or stale bread-and-butter.

DIET FROM TWELVE TO EIGHTEEN MONTHS.

First Meal (7.30 a.m.): A breakfastcupful of milk thickened with groats, fine oatmeal, whole-wheat flour or hominy. Bread or rusks and milk as a change.

Second Meal (about 11 a.m.): A cupful of warm milk, pure or with the addition of one-third lime-water.

Third Meal (1 to 1.30 p.m.): Beef-tea, mutton or chicken broth, thickened with breadcrumbs or sieved potato; as a change, the yolk of a lightly boiled egg. Some milky rice or bread pudding, with the addition of some of the pulp of a roasted apple or the pulp of stewed prunes.

Fourth Meal (5 p.m.): Milk and bread-and-butter.

Fifth Meal (9 p.m.): Warm milk.

DIET FROM EIGHTEEN MONTHS TO THREE YEARS.

First Meal (8 a.m.): Porridge and milk, followed by the yolk of a lightly boiled egg or bread dipped in bacon fat. Stale bread, crisp toast, or a rusk. Milk to drink.

Second Meal (12.30 to 1 p.m.): A little pounded or minced chicken or underdone meat or fish. Milk-pudding and stewed fruit. Water to drink.

Third Meal (4.30 p.m.): Bread-and-butter. Sponge or other plain cake occasionally. Milk.

Fourth Meal (6.30 p.m.): Warm milk and a biscuit.

LECTURE IV

THE DIGESTIVE DISORDERS OF INFANCY—COLIC AND VOMITING

GENTLEMEN,—We come now to the consideration of the disorders of digestion in infancy—disorders which are due, in large measure, to neglect of these rules for the feeding of babies which I tried to bring before you in my last lecture.

I shall not distinguish between the digestive disorders of breast-fed and bottle-fed infants, although for obvious reasons, such disorders are far commoner in the case of bottle feeding than in the case of breast feeding. Further, I shall not attempt to describe these disorders to you from the point of view of pathology or morbid anatomy, because to do so would be to assume a knowledge which we do not possess. I shall deal with them purely from the clinical or symptomatic standpoint.

The symptoms of digestive disorder in infancy are both local and general. The local symptoms are of three sorts—colic, vomiting, and diarrhœa—but I need hardly say that these different symptoms, although one describes them separately, very commonly coexist in the same case. The second or general group of symptoms may be comprised in the one term 'wasting.' To-day I shall deal only with the local symptoms. The general symptom—wasting—may be more conveniently deferred until we speak of marasmus as a special disease, digestive disorders being only one of many causes which may lead to it.

COLIC.

Let us begin, then, with that very common disorder griping, or colic. I think one can distinguish three classes as giving rise to the painful contraction of the intestines which we call colic. The first of these is acidity due to fermentation occurring in the milk and producing lactic and other acids, which irritate the bowel and induce over-contraction in its wall. The second and perhaps the commoner cause is the existence of undigested casein or curds in the intestine. I told you that the great difficulty in the feeding of infants with cow's milk is the indigestibility of the casein and there is no commoner consequence of that than the production of colic. The third cause is the presence of gas in the bowel.

How are you to recognise colic? The chief symptom is pain. Of course, pain in the case of infants is expressed not in words, but by cries, and whenever you find constant and frequent screaming it should always make you think of the possibility of colic, for that is one of the commonest causes of persistent screaming. There are, however, other causes for screaming which may lead you into error. One of these is *earache*. It is often difficult to be sure of the existence of earache in young infants; but in such a case the mother will probably tell you that the child has been in the habit of putting his hand to the ear or head, and this may indicate to you the seat of pain. Another possible cause of persistent screaming is *teething*, a cause which mothers certainly tend to exaggerate, but, nevertheless, it is one which you should always have in mind as a possibility. Teething and earache [illegible] often to go together. Another cause, and one which is commoner than you think, just as [illegible] is less common than you are apt to think, is *renal* [illegible] perhaps surprising to you to be told that little [illegible] suffer from renal colic at all, but the probability is [illegible] suffer from it as frequently as grown-up persons [illegible]. I do not mean to say that children pass [illegible] gravel. You may have seen in the post-mortem [illegible] the kidneys of quite young infants which contained uric acid infarcts, and even in fœtuses *in utero* you may find them. Another possible cause of screaming is *phimosis*, which gives rise to straining on passing water; and the pain which that entails, the result of what we may perhaps speak of as a colic of the bladder, expresses itself in screaming. Another group of causes which you have to think of when a child is constantly screaming is *tenderness of the bones*, resulting from scurvy, or possibly from rickets in its acuter forms, or from congenital specific disease. In that case the screaming will be more noticeable when the child is handled, or when he is put into his bath, and that, along with a careful examination of the bones, will enable you to establish or exclude this cause. There remains one other cause of screaming which you ought to think of when you have excluded every other possibility, and that is *mental deficiency*. One of the signs of defective cerebral development in young infants is screaming without apparent reason. You will, therefore, bear in mind those possible causes before you conclude too hastily that the child is screaming because of colic. But you must also remember that colic is perhaps commoner than all these other causes put together.

The **signs by which you recognise colic** are, in the first place, hardness of the abdomen. You put your hand on the child's abdomen, and feel that it is unusually resistant ; indeed, it may be almost knotty. You may feel little coils of intestine standing out, coils of firmly contracted bowel. And you will observe, in the second place, that as the child screams he tends to draw up his legs. The screaming may even be so extreme that the child passes into a condition of convulsion, for there can be no doubt that the irritation caused by undigested food in the intestines may be an exciting cause of convulsions in young children. Another sign by which you can be quite certain that the pain is due to colic is the cessation of the screaming on the passage of flatus. If that happens you may be sure that colic was the cause.

Those being the signs and symptoms by which you recognise colic, we now come to consider **how you are to treat it.** I shall describe first of all what ought to be the immediate treatment of an attack of colic, and then I shall ask you to consider with me by what means you may prevent the attacks coming back. The first thing to be done in an attack is to apply warmth to the abdomen. You can do that by friction with warm oil, by warm fomentations, or by poultices of linseed or mustard ; and in very extreme cases it may even be necessary to use the mustard bath. In addition to the external application of warmth you can apply it internally with advantage by means of an enema of warm water. Inject 2 or 3 ounces of water, as hot as the child will bear it, well up into the large intestine. In addition it is well to give carminatives by the mouth to aid in the expulsion of flatus ; peppermint-water or dill-water are suitable for the purpose. Some prefer to give small doses of sweet spirit of nitre, say 10 drops.

By means such as these you will generally succeed in cutting short an attack of colic, and then you have to consider how you are to prevent its recurrence. One may divide cases for this purpose into two groups : first, those which are being fed by the breast ; and, secondly, those which are being fed by the bottle. If a breast-fed child is suffering from colic you will probably find on inquiry that it is being fed too often or at irregular intervals. All that you require to do in such a case is to regulate the feeding—to feed every two and a half hours instead of every two hours, or every three hours instead of every two and a half, as the case may be. The tendency to overfeeding in those cases is a natural one, because the taking of warm milk into the stomach, just as the injection of warm water into the rectum, temporarily relieves the colic, and the mother, finding the child is relieved by suckling, tends to go on giving the breast too often, and so a vicious circle is set up ; and what you have to set yourself to do is to break that vicious circle by seeing that the child is fed regularly by the clock, and not at irregular intervals. In some cases the colic of breast-fed children seems to be due to the casein of the mother's milk being either too abundant or for some reason more difficult of digestion than usual. When you meet with such a case you will sometimes find it of advantage to administer a few teaspoonfuls of lime-water to the child before each feed, the lime-water being sweetened a little to make it acceptable to the child. Or you may give a couple of grains of citrate of soda dissolved in a tablespoonful of warm water in the middle of the feed. Further, you will remember to correct any constipation, which is so common in breast-fed children—a most important point, and one which I shall deal with in another lecture (Lecture VIII.).

If the colic still persists, the question will arise, Are you to wean the child or not? Here I would remind you of what I said in the last lecture, that mere digestive disorder is by itself no necessary indication for weaning; it is only if the digestive disorder is accompanied by a persistent and steady loss of weight that you are justified in taking that step. So if, after correcting errors of feeding and constipation, the weight still goes down, you may consider the question of weaning.

We will pass now to the commoner state of things which you have to deal with—namely, the prevention of colic in an infant who is being fed by the bottle. The great cause of **colic in bottle-fed babies** is a want of cleanliness in preparing the milk? and in particular it is the continuous use of those tube bottles of which I have already had occasion to point out to you the dangers. Such bottles produce far more colic, probably, than any other one cause. Your first duty is to see that the child has a proper and clean bottle; and, secondly, just as in the case of breast-fed children, see that the feeds are regulated, and not given too often. Your third care will be to see that the mixture which the child gets is sufficiently digestible. Assuming that the bottle is clean, and, therefore, that fermentative action has not occurred, the colic may be put down to the indigestibility of casein. To correct that you have to give a mixture which contains less casein, or in which the casein has been rendered more digestible in one of the ways I have described when speaking of infant feeding. You may also require, as in the case of breast-fed children, to correct constipation; but more commonly you will have to deal with diarrhœa, and the best ways of treating that we shall consider when we speak of diarrhœa as a symptom of digestive disorder.

In all cases of colic, both in breast-fed and bottle-fed children, you will find the administration of a **carminative** between the feeds of help. A carminative mixture for a young infant should contain certain definite ingredients. First of all, it should be alkaline, and therefore you use bicarbonate of soda (2½ grains) as the basis. It should be alkaline, because colic is so often due to acidity in the intestine. You supplement this with an aromatic mixture which is also alkaline ; for instance, aromatic spirits of ammonia, 2½ minims. Secondly, to make this pleasant, you will add a little glycerine (2 minims). It is always advisable if you can to make medicines pleasant for infants, and one of the best ways of doing this is to add glycerine. Syrupy preparations are less suitable, because they are apt to ferment, and as you have already got fermentation going on you do not want to do anything to increase it. Thirdly, you make the mixture up to a teaspoonful with peppermint or some other carminative water. This should be given between the feeds, and it will be found useful in all cases. In addition, you should see that a child who suffers from colic is kept very warm. A common, but often unsuspected, cause of the disorder is chilling of the abdomen. Inquire, therefore, into the covering of the child's abdomen, and insist upon his having a broad flannel binder, even though he be beyond the age at which binders are used, and see that the binder does not slip up over the chest and leave the abdomen unprotected. See also that the feet and legs are kept warm for cold feet are a very common cause of colic in young babies. The child should have warm socks, and, if necessary, a hot bottle to the feet. If, in spite of all you do, the colic persists, and if at the same time the infant is gaining weight (which sometimes does happen), so that you are not justified in making any great alteration in the feeding, you may perhaps require to have recourse to opium in some shape. You should not give opium any oftener than you can help, but sometimes you are driven to use it owing to the persistent pain which the child suffers. The best preparation to use is codein, $\frac{1}{40}$ or $\frac{1}{30}$ grain, which, with glycerine and some carminative water, makes a very suitable opiate for a young child.

VOMITING.

I now pass on to the consideration of *vomiting* as a sign of dyspepsia in young infants. There are two varieties of vomiting which you will learn to recognize when you get into practice as signs of digestive disorder. There is (1) *acute vomiting* which is usually the result of an attack of gastric catarrh; and there is (2) *chronic vomiting*, which may be the result of chronic gastric catarrh, but is very commonly due merely to unsuitable feeding.

You will have no difficulty in recognizing the **acute type.** The child will be brought to you with the history that he began to vomit a few hours before. You will find that the skin is hot, the tongue thickly furred, and that there is a sour smell about the breath. The only difficulty in your diagnosis will be the exclusion of more serious disease. Many of the acute specific fevers in children begin with vomiting; so do many intracranial diseases, and you may at first be doubtful whether you are not dealing with some such case, particularly as acute gastric catarrh is often accompanied by a high degree of pyrexia. You may do wisely, therefore, to reserve your diagnosis at first.

The proper treatment of acute vomiting is by **starvation.** You need never be afraid to starve young children in the matter of solid food. It is said that infants bear starvation badly. That may be true of chronic underfeeding, but babies will stand the complete withdrawal of all nourishment for two or three, or even more, days without any disadvantage, and, indeed, often with great benefit, provided always that you fulfil two conditions. The first of these is that the child is kept warm. Children stand starvation badly simply because they lose so much heat from their surfaces, a fact which is true of all young animals. And the second condition is that water must on no account be withheld. Children stand the withdrawal of fluid very badly indeed. So when I say that they are to be starved it means that they are to be given nothing which can be described as nourishment, but they must have abundant liquid; and in such cases as we speak of that may take the form of boiled water, or, if you like, and in order to satisfy the mother, one may administer a thin decoction of barley or weak veal broth. The liquid, whatever it may be, should be given cold with a teaspoon.

You will find, also, a great help in these cases from **washing out the stomach.** This is the first time I have had occasion to mention washing out of the stomach as a therapeutic measure in children. I can only say that you will find it of the very greatest use in all cases, acute or chronic, whether in breast-fed or bottle-fed infants, where vomiting is a prominent symptom. The method is so simple, it is so safe, and, compared with a similar proceeding in adults, so comparatively pleasant to the patient, that you need have no hesitation in employing it. You should use for the purpose a small-size red rubber œsophageal tube. The œsophagus in a baby is bigger than you might think ; it is certainly bigger than an ordinary pencil, and you will be able to get the smallest size of tube down it without difficulty. It is an advantage to use a tube which is rather large than otherwise, because the tendency of a small tube is to permit regurgitation of fluid alongside, which leads to coughing and choking, and to struggling on the part of the child. The œsophageal tube is connected by means of a piece of glass tubing to another piece of soft rubber tube, and that to a funnel such as I show you, which is really nothing more than the barrel part of a glass syringe from which the piston has been removed. A funnel of this shape

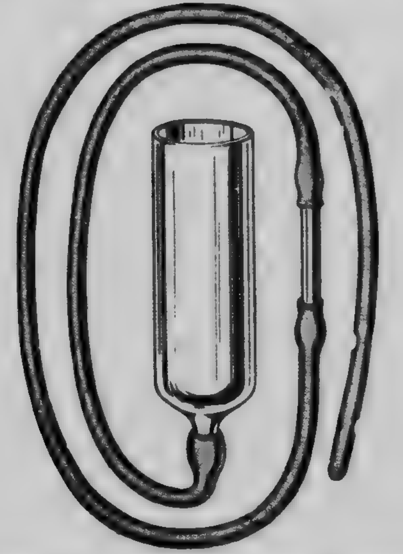

Diagram 8.—Stomach-Washing Apparatus.

has the advantage that you can blow down it if you want to. Sometimes the eyelet of the œsophageal tube gets blocked with a mass of casein, and it is then an advantage to be able to put your mouth over the top of the funnel and displace the casein by blowing. By the same means you can produce a sort of shower-bath in the interior of the stomach, and by projecting the fluid with some force through the tube you can wash away the mucus and masses of casein which have accumulated there. It does not matter much what fluid is used in the washing out. I usually employ plain water. You can use it quite hot, remembering that it always tends to get cooler as it passes down. But if there is a very sour odour in the breath, I recommend you to add a little bicarbonate of soda—a teaspoonful to the pint—before the final washing. Having completed the washing, you will find it an advantage to leave an ounce or two of fluid behind, for this helps to fulfil the condition which I have laid down, that the child must be kept supplied with a large quantity of liquid when he is being starved. The hot water also acts as a kind of internal poultice, and is soothing. After this manipulation a child will often go quietly off to sleep, though he may have been screaming for some hours before the washing out was done.

You will not find **drugs** of much assistance **in acute vomiting.** If you wish to have recourse to them, one would recommend especially small repeated doses of calomel, particularly if the tongue be much furred ; $\frac{1}{10}$ to $\frac{1}{6}$ grain every hour for half a dozen doses is a suitable quantity for an infant, and that may be followed by the use of bismuth, either the carbonate or the subnitrate. Be sure always to give bismuth in large doses ; that is to say, never less than 10 grains, and even in a young infant you can give more than that with advantage.

The **chronic vomiting** which is the result of digestive disorder is of quite a different nature to that I have been describing ; and I would make this first remark about chronic vomiting in young infants, *that it is of no importance at all unless it be accompanied by progressive loss of weight.* You are not to get anxious, as nurses and mothers do, because a child vomits after his feeds. Many children, like the Romans of old, have a way of swallowing more than they want and rejecting the excess, and that is not a habit which need alarm you. Indeed, there is a saying amongst the nurses that 'sick babies always thrive.' It is only when the vomiting is accompanied by progressive loss of weight that you should take it seriously. The second remark I would make is, that if you are dealing with quite a young infant—that is to say, one a few days, or at most a few weeks old—and if the vomiting has dated from birth, you should make certain if you can that you are not dealing with a case of **congenital pyloric obstruction.** That, however, is a subject of so much importance that I shall devote a special lecture to its consideration.

Now, **how should you treat** that **chronic** form of **vomiting** which is the result of purely digestive disorder ? The first thing is to regulate the feeding. Vomiting, if it is habitual, means that the child is getting a mixture which is too strong for it, and you must dilute the milk still further, or else give it in a more digestible form. And here, again, it will be extremely important to regulate the times of feeding, taking care that the bottle is not given too often. In these cases, also, you will find periodic washing out of the stomach to be of the very greatest service. You can employ it every day, or even oftener, with the greatest benefit. With regard to drugs, I would only say again that they must take quite a subsidiary place. It is only after you have corrected the feeding and washed out the stomach thoroughly that you should think of drugs at all. You will find bismuth one of the most useful if given in large doses. In other cases, again, 2 or 3 minims of dilute hydrochloric acid, with a little glycerine and some carminative water, will be advantageously prescribed. I cannot tell you beforehand which cases will do best with bismuth and which with hydrochloric acid. I think it is more or less an experiment in every case ; but one or other of these drugs will generally be very beneficial, supposing you have failed to relieve the child by adjusting the feeding and washing out the stomach.

LECTURE V

CONGENITAL PYLORIC STENOSIS

Gentlemen.—Congenital hypertrophy of the pylorus, or congenital hypertrophic stenosis, as it is sometimes termed, the subject of my lecture to-day, is a condition which has only been generally recognized in the last decade or so, but which is probably very much commoner than is generally believed, and I am perfectly sure that anyone who is capable of diagnosing it will meet with cases in his practice not very infrequently. Inasmuch as the treatment of this condition, if it is to be successful, must be begun early, it is extremely important that you should be able to diagnose it when you see it, and that is why I am devoting a special lecture to it.

I shall not trouble you with the history of our knowledge of congenital pyloric stenosis, because that really does not throw any particular light upon the nature of the condition, and it is of no use to you in your practice, so I shall begin at once with the **symptoms,** which, fortunately, are few and definite. The kind of case which exhibits congenital pyloric stenosis is this : The child is usually—indeed, I may say nine times out of ten—a boy, and he is brought to you when a few weeks old because, as the mother says, she cannot find any food to suit him at all. She will complain that, no matter what food she tries, it results in the production of vomiting. You will usually find that a great many different foods have been tried—many of the patients, indeed, have run the whole gamut of the patent infant foods in the market—and yet the vomiting goes on. There is a very curious fact about the vomiting, and it is this : that when a new food is tried it will often seem to suit the child for a period of about twenty-four hours, or a little longer, and then the vomiting begins again, and will go on every day until a new food is tried, when it will again often cease for a day or two, only to recommence once more. At each of these occurrences the hopes of the mother are buoyed up ; she believes she has at last found the kind of food that is going to suit ; but they are immediately dashed to the ground again when the vomiting reasserts itself. Why it is that the new food is often borne for a time in these cases I cannot tell you ; it is one of the many curious clinical facts that characterize this condition.

On careful inquiry, you will usually find that the vomiting has not strictly dated from the moment of birth. As a rule, you will be told that the child was born a fine, healthy child ; that he was fed from the breast at first, and all went well for a week, ten days, or a fortnight, but then vomiting began, and the child was taken off the breast, very often by medical advice, because of the continuous sickness, and put on to some artificial food. As that was also followed by vomiting, another artificial food was tried, and so on.

I want you to observe—because it has some bearing upon the view that one takes of the pathology of the condition—that the vomiting is not, strictly speaking, congenital ; it is more usual for it to begin ten days or a fortnight after birth. In addition, the vomiting has certain peculiarities which characterize it and distinguish it from the ordinary vomiting of dyspepsia. In the first place, it is often extremely violent, or what is termed 'projectile'—that is to say, the child will suddenly throw out the stomach contents forcibly, often projecting them quite a long way from itself. This forcible or projectile character is one of the things that distinguishes the vomiting of congenital pyloric stenosis from that which is due to ordinary dyspepsia. The vomiting is also characterized by its abundance, and the mother will tell you that the child brings up much more food than it swallowed at its last feed. Further, you will often be told that the child brings up a quantity of 'slime' with the vomit. Slime is what you and I know as mucus, and the bringing up of a quantity of mucus is another of the things which characterize this particular kind of vomiting.

Another prominent symptom that the child will exhibit besides the characteristic vomiting is *progressive loss of weight*. That, of course, is natural. If the child is constantly vomiting, it naturally loses weight, and along with the loss of weight there is a shrivelling up of the skin, and a loss of its elasticity, such as you meet with in cases of chronic diarrhœa in children. The loss of weight may proceed until it reaches a very serious degree ; such children may waste away until they are literally mere skin and bone.

The third thing which characterizes a case of pyloric stenosis is that the child is usually—indeed, almost invariably—*constipated*. That is a sign of very great importance, because if a child is vomiting from unsuitability of food, instead of being constipated, it usually has diarrhœa ; and a combination of vomiting *plus* constipation should always arouse in your minds strong suspicions that you are dealing with a case of this sort. The reason for the constipation in this case is obvious. If no food, or very little food, is entering the intestines, obviously there must be very little in the way of excreta to go out of the intestines, and the constipation, therefore, is what is sometimes called the 'constipation of starvation.' It is the inevitable result of the small amount of nutriment which is finding its way through the pylorus into the bowel.

When you come to examine the child for **physical signs**, you will be at once struck by the *emaciation*. Observe that, although these children are emaciated, they are not cachectic, and that is a point worth noting. They show the thinness of starvation, but not the thinness of cachexia. In other words, although thin, often miserably so, they are not anæmic, and you know that it is a combination of emaciation with anæmia which makes up cachexia.

In examining the abdomen, you will usually find that there is a certain amount of fulness in the upper part above the umbilicus, whereas the rest of the abdomen is sunken. And then, having your suspicions aroused, you should look for what is, after all, the most characteristic sign of the condition—viz., *visible stomach peristalsis*. If you are going to succeed in seeing visible stomach peristalsis, you must look for it when the stomach is full. There is no use in

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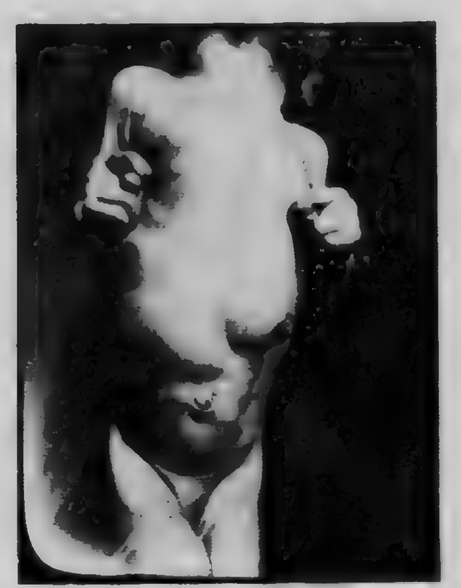

FIG. 4.

VISIBLE STOMACH PERISTALSIS IN PYLORIC STENOSIS.

[illegible]

examining a child for gastric peristalsis shortly after it has been sick ; if you wish to make it out, you should always insist upon the child being fed in your presence, and immediately it has taken its food you should make your examination. If the peristalsis be not immediately visible, you may sometimes elicit it by applying friction over the stomach ; massaging the stomach may bring it out. This visible peristalsis assumes a very characteristic form. You will see appear below the left costal margin a swelling about the size of a golf-ball, which will begin to travel downwards to the right, sweeping above, or even over, the umbilicus, and which, before it has disappeared, is followed by another swelling, so that a dumbbell or hourglass-like appearance is produced over the upper part of the abdomen. Sometimes in extreme cases you will see three such swellings —or, in other words, three waves of peristalsis—simultaneously visible over the stomach. Such peristalsis, *if it be well marked*, is absolutely pathognomonic of the condition, and sometimes it is extremely well marked. I have seen it so well marked that the swelling of the stomach could be seen through the child's clothing. Unless it be well marked you should not attach importance to it, because *slight* gastric peristalsis may be visible as a purely transitory phenomenon in circumstances other than those in which there is an actual constriction of the pylorus.

In addition to the emaciation and the visible peristalsis, you will find in many, but by no means in all, of these cases that you can actually feel the *thickened pylorus ;* and you can feel it usually to the right of the umbilicus. Observe that sometimes the pylorus cannot be felt, even in undoubted cases, for the simple reason that it lies tucked up under the liver where you cannot get at it. But if you wish to give yourself every chance in feeling for it, there is a particular way of setting about it, and that is that you should not merely press downwards and backwards as one does in ordinary palpation of the abdomen, but you should pinch up the deep structures, and try and grasp the thickened pylorus between your fingers and thumb. You then find an elongated swelling about the size of a hazel-nut, or a little larger ; and sometimes, if you are lucky, you will feel it actually undergoing contractions whilst you feel it ; you feel it undergoing relaxation, followed by hardening.

If you proceed further in your examination by washing out the stomach, you will usually be able to make out that the capacity of the stomach is increased—that instead of holding, let us say, a couple of ounces or so, which is normal for that time of life, the stomach holds 3, 4, or even more ounces of fluid. In other words, you can prove the existence of actual *gastric dilatation*, and that, in addition to the peristalsis and the palpable tumour, is the third purely stomach sign of this condition. So much, then, for the symptoms and signs.

It will be well for us now to look for a moment at what is known as the **pathology of congenital pyloric stenosis,** and I may say at once as regards this subject that we do not know what the real pathology of the condition is. What we do know is that in those cases which prove fatal, or in which operation has been performed and an opportunity has been given of inspecting the pylorus during life, there is present a great thickening of the pylorus, which section shows to be due almost exclusively to hypertrophy of the circular muscular sphincter of the pylorus. I have here a few specimens which show this thickening very well. Here, for example, is one in which you can see quite well with the naked eye the ring of thick muscular tissue surrounding the pylorus, and which practically converts it into a tube of almost cartilaginous consistence. That is one thing you can make out. Another thing that you can find is that the whole wall of the stomach is more or less hypertrophied owing to the efforts of the stomach to drive its contents through this narrow channel. Thirdly, you will find that the mucous membrane of the stomach is in a state of chronic catarrh. That summarizes all there is to be said about the morbid anatomy of the condition : there is an hypertrophy or overgrowth of the circular fibres of the pylorus there is a thickening of the whole wall of the stomach, and there is catarrh of the mucous membrane.

So far everyone is agreed, but when you come to attempts to explain how these anatomical findings are produced you at once enter the sphere of very acute controversy. You may say that, roughly speaking, there are two views prevalent as to how it is that this condition is brought about. There is, first, the view that *the hypertrophy of the muscular fibres of the pylorus is a congenital anomaly*—that there is a true overgrowth, a true hypertrophy of the circular muscular fibres existing as a congenital anomaly, just as you may have a congenital club-foot or any other inborn malformation. The other chief view of the pathology of the condition is that the hypertrophy is not a congenital malformation at all, but is *consecutive to a spasm of the pylorus.* Those who adopt this view maintain that the condition of which we are speaking is, in its essence, a spasm of the plyorus of great severity and of long continuance, and that, as a consequence of the continued spasm, you get what you obtain from over-use of muscle anywhere—hypertrophy—and that that thick pylorus which I showed you in the specimen is simply the result of over-use—it is the result of prolonged and repeated spasm of the sphincter of the pylorus. When those who hold this view are asked how this spasm is brought about, they are compelled to have recourse to various hypothetical explanations. Some assume that there is an 'inco-ordination.' Now, gentlemen, inco-ordination is one of those expressions like 'trophic effect,' and 'toxin,' and 'reflex action,' and a few others which are often used as a cover for much ignorance; and it is no more satisfactory in this case than it usually is. Then there are some who say the spasm of the pylorus is brought about by an excess of acid in the stomach contents, because it is known that excess of acid in the stomach leads to the closing of the pylorus. No chemical proof has been advanced that such excessive acidity actually exists. Others say that something has gone wrong with the chemical mechanism in the duodenum, which is really responsible, as Pawlow has shown, for the normal opening and shutting of the pylorus. But all these explanations, I confess, are extremely unsatisfactory. My own view is that the condition is not a congenital anomaly, but that the second view is right, explain it how you may, and that we have here primarily to do with a spasm. In favour of that I would only point to the two great clinical facts which cannot be got over: first, that the symptoms do not come on immediately after birth, but generally a fortnight or more later; and, second, that the condition is capable of being absolutely cured by purely medical treatment, leaving the stomach, as I have proved to my own satisfaction time and again, in a perfectly normal condition afterwards. I cannot see, on the assumption of a congenital anomaly of formation, how such cases should make a perfect recovery without operation and without any dilatation of the stomach being left. I believe, therefore, that we have here to do with a spasm which goes on to hypertrophy, but how that spasm is brought about I confess I do not know, and from the point of view of treatment I do not think it matters.

That brings me to speak of **treatment**. Now the first essential here is that you should begin treatment early. I believe that failure in these cases is due to the condition being recognized too late, with the result that treatment is left until the child is in such an exhausted condition that recovery is difficult, if not impossible. That just emphasizes what I have already said about the importance of early diagnosis.

The first and most important of all things in treatment—I put it before diet and everything else—is systematic *washing out of the stomach*. That is *the* main factor in getting these cases well. You wash out in the ordinary way every day, or twice a day if need be. I do not think it much matters what you use to wash out the stomach with, but at first there is an advantage in using a weak alkaline solution—a weak solution of bicarbonate of soda, for example—because there is often so much mucus in the stomach owing to the prolonged catarrh of the mucous membrane, which has resulted from stagnation of the contents, that it is an advantage to use an alkaline wash to bring the mucus into solution, and so get rid of it. I prefer, therefore, at the outset to use a drachm of bicarbonate of soda to a pint of warm water, and use it once or twice a day. The immediate result of the washing is that the vomiting ceases, or almost ceases; it will cease almost as soon as you have got rid of the mucus.

The second question that faces you in treatment is, *how you are to feed the child*. Your object must be to give food which will pass through the pylorus as easily as possible, and therefore you will select a milk which does not form large clots—that is obvious. As a matter of fact there is nothing better, when you can get it, than human milk, and that is why it is unfortunate when these children have been weaned early, as they usually are, under the impression that breast milk is disagreeing with them. That is always a pity, because you will find when you wean them that you are jumping out of the frying-pan into the fire, for if these children are not able to retain human milk the chances are they will not retain anything else. So that if the child is at the breast, you should keep it at the breast ; if it is off the breast, it is a good plan to get a wet nurse. The practical difficulty of breast-feeding is that these patients need to be fed frequently and in small quantities, and that becomes a nuisance to the mother. In some cases you may have to draw off the breast milk by a pump, and feed the child with it from a bottle or by teaspoon at short intervals of, say, every hour. Assuming that human milk is unobtainable, I believe the next best thing is fully peptonized milk, given diluted with an equal quantity of water, and I do not believe that, beyond that, there is any good in searching about for a particular food that will suit these children. If you adopt lavage, and do it properly, you will find that they will retain their food well enough, but you will never find anything which will pass quite satisfactorily through the pylorus, at first at least. Hence, although there are many combinations on which these patients will do well, such as whey and cream, whey and egg-white, desiccated milk, malted milk, and so on, yet my experience has been that if they do not do well on peptonized milk they will not do better on anything else, and there is no use in perplexing yourself by a large choice of methods of feeding.

As regards the intervals of feeding, the frequency and the quantity, you have to be guided by the capacity of the stomach in the particular case with which you are dealing ; you have to feel your way, and to notice how much the child can stand without being sick. Sometimes you have to feed every half-hour, or every hour or two hours. Here again you will find that, once you start washing out, you will soon be able to get the child down to approximately the ordinary intervals which are natural and right for a healthy child of that age.

Third, as regards the question of *drugs*, I believe there is none which is of any real use in this condition. Opium in minute doses has been highly recommended, $\frac{1}{20}$ to $\frac{1}{40}$ minim of the tincture given shortly before each feed. These quantities seem almost infinitesimal, almost homœopathic, and yet I have known one of these children rendered almost comatose by $\frac{1}{20}$ minim of the tincture of opium given before each feed for less than a day. Owing to their exhausted and feeble condition, they are extraordinarily sensitive to opium, but some, particularly German, writers have reported very great benefit from giving it, the theory being that it helps to abolish the spasm of the pylorus. Although I see no objection to trying opium, personally I have been disappointed in the results it has yielded. I have tried alkalies in a few cases, and I have tried belladonna, which has a reputation as an antispasmodic, and in neither case have I found any benefit from so doing. The treatment, therefore, resolves itself into careful feeding and nursing, and persistent lavage of the stomach. You will be told sometimes that it is an advantage in these cases to feed *per rectum*, or, rather, to give water or saline solution *per rectum*, in order to make up the deficiency in fluid in the body which undoubtedly exists. As to that I would only say that my experience has been that it is almost impossible to get children of that age to retain saline enemata for any length of time, and, therefore, although such treatment is perfectly right in theory, in practice it is almost impossible to carry it out.

You may say, What are the **results of this treatment?** I can only tell you that, taking my own experience of these cases in private practice, and confining myself to well-marked cases, about the diagnosis of which there could be no reasonable doubt—cases in which the gastric peristalsis was *very* well marked, in which there was great emaciation, and in many of which, at least, though I admit not in all, a thickened pylorus could be felt. I have had twenty which were treated medically, and only two of those died. I have followed these cases out, some of them for three or four years, and they have remained perfectly healthy children. The question is, Are they left with any dilatation of the stomach ? I would say that, in the majority of cases, so far as I have seen, they are left with no dilatation of the stomach ; the condition seems to be one of genuine cure. But every now and then you will meet people in adult life who have stomach symptoms, signs of dilated stomach particularly, who date their symptoms right back to childhood, and who are, I believe, cases of imperfectly recovered congenital pyloric hypertrophy. I saw last week a young lady whose gastric symptoms went right back to childhood. Her mother told me that when she was two months old she was reduced to a skeleton, and was constantly sick ; they could not get a food to suit her. She ultimately recovered, but she has suffered more or less from dyspepsia ever since, and at the age of five it was noticed that her stomach was dilated. I believe that this was a case where one had to do with congenital hypertrophy of the pylorus, which had not completely disappeared. But that is an exceptional occurrence ; the great majority of those I have seen do get perfectly well. I do not profess to explain how it is that such treatment cures those cases ; I only say the fact that they are cured is, to my mind, an argument that it is a condition of spasm and not of true hypertrophy, and that if you can get rid of the spasm the muscle will go back to its normal size.

There are certain things you must be prepared for in the course of your treatment. I told you that almost as soon as you begin lavage the vomiting will cease, but you must not expect that as soon as the vomiting ceases the child will start to gain weight. Quite the contrary is the case. You should warn the parents that these children will often, after they cease vomiting, go on losing weight, and even to a large amount, for a considerable time. What happens, in my experience, is something like this : The vomiting stops, and yet the weight continues to go down, and down, and down ; everybody gets alarmed, and begins to talk wildly about the necessity for operation. You must keep calm, and say it is all right, that it is the natural course of things. The child goes on losing weight until it is four and a half or five months old, and then, as Browning says, 'Sudden the worst turns the best to the brave, the black minute's at end,' it is round the corner almost at once, and the weight begins to go up. A very experienced hospital sister, who has seen a lot of these cases, once told me that she preferred those children who went rapidly downhill. On one occasion she advocated the child being sent out in order that it might come back much worse, 'because,' she said, 'when they get to a certain pitch of badness, then they generally get round the corner.' It almost seems that when they get to a sufficient degree of weakness the spasm passes off, and it is because people are not aware of this natural course of the disease, even without treatment, that operation is advocated. You may ask, What is your view as to operation? My view is that operation is only rarely to be recommended. I am strongly convinced that if one were to operate on all of these cases as soon as diagnosed the mortality would be much higher than if one were to treat all of them medically. I admit, however, that there are a few cases which may be saved by operation which would succumb if treated on the lines I have recommended to you. The difficulty, however, in practice is to recognize such cases early enough. For of no case of pyloric stenosis can it be truly said that it is so bad that recovery without operation is *impossible*, for I have seen the worst cases get well when left alone. If, on the other hand, one waits too long, it is obvious that one does not give the surgeon a fair chance. The only practical guidance I can give you in this dilemma is to consider seriously the advisability of operation in cases in which (1) the symptoms have set in very soon after birth, and (2) in cases in which, in spite of careful medical treatment carried out for two or three weeks, the child still continues to vomit and to lose in weight and strength. The decision is always a very difficult one, and involves grave responsibility, but it is your duty to make it, for you cannot fairly leave it to the parents.

The exact nature of the operation to be performed and the relative merits of stretching of the pylorus, pyloroplasty, and gastro-enterostomy are surgical questions with which I do not feel competent to deal.

LECTURE VI

DIARRHŒA

GENTLEMEN,—In this lecture I wish to discuss with you the third of the symptoms of digestive disorder which I have already mentioned—namely, *diarrhœa*. I cannot exaggerate the importance of this disease as a scourge of infant life. Recent returns of the Registrar-General show that out of 22,000 deaths from diarrhœa, taking the country all over, 17,000 occurred in children under one year old. That shows you the frequency of this disease, and to what a tremendous extent it destroys life. And you will naturally ask, Why are children so liable to it? I think there are three causes which render children particularly liable to suffer from diarrhœa. The first is that the sterilizing power of the child's stomach is very small; it secretes but little hydrochloric acid, and organisms which are swallowed with the food stand a good chance of running the gauntlet of the stomach and setting up irritation in the intestine. The second is that children are fed mainly upon milk, which is anything but a germ-free fluid; and the third is that infants are particularly liable to chill, for the reasons I have already mentioned. These are the chief causes which render children subject to diarrhœa, and against these you must fight if you wish to prevent it.

Many of the severest forms of diarrhœa in young infants are probably due to specific micro-organisms. Take what is called *epidemic diarrhœa*, which is so common in the summer. That is almost certainly due to a specific microbe. Attempts have been made to isolate it, without complete success, but it is believed that it will prove to be identical, or almost identical, with the bacillus which is the cause of epidemic dysentery. The growth of these micro-organisms is favoured by certain quite definite conditions. If you trace the admissions of acute diarrhœa in such a hospital as this in the summer, you will find that gradually as the weather gets hotter the number of such cases increases, but you will also observe the curious fact that for some time after the thermometer has fallen the admissions still continue numerous; in other words, the curve of epidemic diarrhœa follows approximately the ordinary curve of the thermometer, but not quite accurately. And if you investigate the matter still further, you will find that what it does follow pretty accurately is not the temperature of the air, but the temperature of the soil. It is when the thermometer, immersed 4 feet in the soil, begins to record a temperature of about 56° F., that epidemic diarrhœa becomes most common. In other words, this organism, whatever it is, grows best in the soil about that temperature. Another cause which greatly favours the development of diarrhœa is the existence of bad surroundings—exhalations of all sorts, foul drains, contaminated soil, overcrowding, dirty cesspools, dung-heaps. All these things contribute greatly to the production of diarrhœa; and the importance of these causes is so great that often in practice you will find it an advantage to remove a child, if he is suffering from diarrhœa, right away from his surroundings altogether, and in particular to keep him out of large towns. Further, you will be able to do a great deal in the prevention of diarrhœa by seeing that the milk is boiled before the child gets it, at least in hot weather. You will remember I told you that the importance of boiling milk is to some extent exaggerated. I wish now to qualify that statement by saying that the importance of it cannot be exaggerated in hot weather. All milk given to children in the summer should be boiled.

The third way in which you will be able to do something to prevent diarrhœa is by guarding against chill—seeing that the child is properly clothed, and setting your face against that foolish practice termed 'hardening,' which some mothers will try to get you to favour in the case of their offspring.

By such means as these you will be able to do a great deal in the **prevention of diarrhœa.** But in spite of all you do, there is no doubt you will have a great many such cases in your practice, and therefore we may now pass on to a closer study of this disease and of the treatment appropriate to it.

In classifying the **varieties of diarrhœa** met with in infants I shall proceed upon clinical rather than upon strictly pathological lines, and I shall divide the cases—first, into those which are acute ; and, secondly, into those which are chronic. I shall distinguish among the acute diarrhœas three varieties—those which are simple, those which are febrile, and those which are choleraic—and I shall make no subdivision of the chronic diarrhœas at all. When we come to treatment we shall take both acute and chronic cases together.

If the diarrhœa be acute it may be simple—that is to say, it may be diarrhœa and nothing else ; or it may be accompanied by pyrexia, when we speak of it as febrile ; or it may be so severe in its symptoms, and accompanied by such profuse watery evacuations, that it is described as choleraic. The most dangerous form, what is called epidemic diarrhœa, the familiar 'D. and V.' of the hospital receiving-room, is usually febrile in type. Sometimes in very severe cases it may be choleraic. Of course, this classification is not by any means hard and fast. Diarrhœa cannot be definitely marked off into different classes ; the varieties pass into one another. A case which begins as a simple diarrhœa may become febrile, and from that may become choleraic, just as it may be choleraic from the first, or may begin with fever. Or, on the other hand, it may remain simple throughout. You cannot always refer a case exactly to one or other of these classes.

What are the **symptoms** of infantile diarrhœa ? In the first place, it may begin either gradually or with great suddenness. It may come like a bolt from the blue, affecting a child which was, or appeared to be, in perfect health. The motions are noticed to be more frequent, and often there is more or less vomiting. At first the stools are natural in colour—that is to say, more or less yellow—but before the diarrhœa has lasted long they become greenish, and finally they may come to contain mucus, or even blood.

If the diarrhœa still persists, the motions become extremely offensive and of a watery consistence. I wish, at this point, to say a word or two about the **character of the motions** in different cases of diarrhœa, because I know it is a matter which will be apt to give you some trouble when you get into practice. What is the significance of the different kinds of stools which you meet with in the diarrhœas of infancy? First about the green stools, which are so common and which resemble in appearance chopped parsley or spinach. What is the cause of their greenness? It has been supposed by some that the colour is due to the growth of a special micro-organism which produces a peculiar pigment. I do not wish to deny that that may sometimes be the case; but in the majority of instances I think it is merely due to the bile pigment being hurried on through the intestines without having undergone the usual changes. What is the meaning of the motions being stinking and putrid? That means that peristalsis in the upper part of the tract is too rapid. The consequence of this is that the food is hurried on before it can be properly digested and absorbed. You get, therefore, a mass of imperfectly digested and incompletely absorbed food lodged in the colon, and putrefactive organisms grow in it; hence the putrid stools. There is another variety of stool, which may be described as lumpy or cheesy, where you see white particles, varying in size, more or less oval or rounded in shape, scattered all through the fæcal matter. What is the significance of that? These round or oval particles are the remains of undigested milk; they consist partly of casein and partly of a soapy substance formed from the fat of milk and lime salts, and they indicate that the child is getting more milk than he can digest. Then there is a kind of stool in children which is extremely irritating, and which scalds the skin. If a child has that form of diarrhœa he gets an erythematous eruption over the buttocks, and I have seen those stools so irritating that a drop of them falling on the dorsum of the foot produced a blister in a few minutes. To what do these stools owe their irritating properties? It is due, I think, to the development of fatty acids—butyric acid and its allies—and means that there is too much fat in the diet. Excess of sugar, on the other hand, produces frothy acid motions of natural colour. Then there is a stool which you may describe as 'slimy'; that means a stool which contains mucus in excessive quantity. Remember, however, that barley-water may produce a slimy-looking stool, which looks very like one which contains mucus. If you recognize excess of mucus, you are justified in believing that the large intestine is particularly involved; and if blood is passed in the motion, as it sometimes is, that suspicion amounts to a certainty. Such is the significance of the chief varieties of stools which you will meet with in the diarrhœas of infants.

We will now pass on to look at some of the other symptoms. The **general symptoms** may be trivial, as they usually are in simple diarrhœa; or they may be extremely severe, as they always are in the choleraic form. Where the constitutional symptoms of diarrhœa are severe, you are justified in concluding that you are dealing with bacterial poisoning. There can be no doubt that the extreme collapse and depression from which those children suffer is not the consequence merely of the draining away of fluid from the body, although that helps in their production, but they are a direct result of poisoning by the absorption of toxins from the alimentary canal. Those symptoms may be described as the symptoms of collapse. The fontanelle becomes depressed, the eyes sunken and staring, and by-and-by they are kept partially open while the child is asleep, and you can perceive mucus on the cornea. The skin becomes wrinkled, the child seeming, as it were, to shrink away from his skin, which becomes redundant; and when you pinch it up, instead of falling back again as it would normally, it is apt to remain in folds. When you get this condition, which is described as a want of elasticity in the skin, you can be certain you are always dealing with a severe case. It is one of the points always to be looked for in your cases of infantile diarrhœa. Many observers believe that when such a condition of the skin is present you are dealing with a case in which there is an involvement of the kidneys, and in which partial suppression of urine is taking place. And unquestionably, if you examine the urine in those infants,

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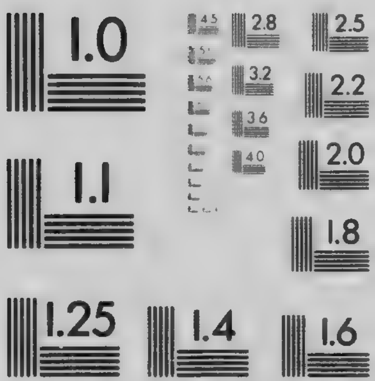

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you will usually find that it contains some albumin. The importance of recognizing this want of elasticity in the skin is based upon the fact that when you find it you should institute special means of treatment, which I shall describe to you later. If the diarrhœa goes on, the symptoms of collapse become more pronounced. The temperature, which was at first perhaps high, falls, at least on the surface of the body, though it may remain high in the rectum ; the child becomes blue and livid, and finally dies, death being often preceded by convulsions. That is the usual picture of a typical case of diarrhœa of severe type in a young child. I need hardly say, however, that the picture varies a great deal. In the simple form there will be little evidence of collapse at all ; in the choleraic form the signs of collapse will dominate the whole scene ; whereas in the febrile cases the depression may not at first be very great, but becomes more marked at the close.

We may now pass to the **treatment of acute diarrhœa.** Here, gentlemen, I would wish to give you this piece of advice : Put not your faith in drugs. Your watchwords in the treatment of acute diarrhœa in an infant must be two—one is *starvation*, the other is *elimination*. If you bear in mind these two great indications, you will never go far wrong in the treatment of your case.

A word about each of those indications. You must **starve the child** because you do not wish to furnish any further pabulum for the growth of micro-organisms ; and milk in particular you must withhold, for there is reason to believe that milk in such cases is actually poisonous. In a case of acute diarrhœa, whatever you do you must stop milk. With regard to elimination, your idea should be, in the first place, to remove as far as you can those organisms which are still growing in the alimentary canal, and, in the second place, to get rid of their poisons.

How are you to carry out those indications in practice ? *Starvation* is easily carried out by withholding food ; but, as I have told you before, infants only bear starvation well if you keep them warm and at the same time supply them with plenty of liquid. Withhold all food for twenty-four hours if there is severe vomiting, and give sips of boiled water, or a weak decoction of barley-water or rice-water. Or, if you want to make a pretence of giving some nourishment, give a weak veal broth or raw egg-white mixed with water, one or two teaspoonfuls at a time. Even in that case, however, I think the benefit is derived from the water. I think it is a matter of indifference whether you put anything in it or not. Still, as I say, you may wish to defer to the wishes of the parent in the matter of appearing to give something nourishing. If the case is not doing well you can continue starvation for two or even three days. As the symptoms abate you may begin to give a little nourishment in the shape of whey, but the thing which you should give last, and be most careful about giving, is the casein of milk, because that is what does harm more than anything else. You may add raw egg-white to the whey, and continue this mixture until the motions are satisfactory.

With regard to *elimination,* if there is vomiting, begin by **washing out the stomach** in the manner I described in my last lecture. If the diarrhœa be severe, and especially if there is reason to suppose that the large intestine is chiefly involved, **wash out the colon** also—that is to say, begin your treatment by washing out 'at both ends.' I have already described to you the technique of washing out the stomach, and I would like now to say a word about washing out the bowel. I do not think it matters much what solution you use for washing out. Warm saline is as good as anything else. It should be given through a douche-can or funnel. Care must be taken not to hold the can too high ; it should never be raised more than 2 feet. Remember it is not difficult to rupture the colon of a child in attempts to wash out the bowel. I think it is best to have the child lying on his back, with the hips slightly raised, so that the fluid can gravitate down into the colon, using a small-bore soft œsophageal tube, just as for washing out the stomach. The tube should be introduced carefully, and you will find its introduction much more easy and satisfactory if you keep the fluid flowing all the time. The reason for this is, as you will readily see, that the colon being only a potential cavity, and its walls more or less in apposition, the liquid as it runs in clears a way for the tube, and so you are less likely to get the end of it entangled in folds of mucous membrane, or to push it through the wall of the bowel. Pass the tube up as high as you can—you cannot pass it too high. Irrigate the intestine thoroughly, until the washings come away clear. By so doing you will wash away some at least of the organisms which are growing there. You also wash away a certain amount of the toxic products which these bacteria have been producing.

In order to favour elimination you should have recourse in all severe diarrhœas to **subcutaneous injections** of fluid. In cases accompanied by collapse, especially in those in which the skin is inelastic, elimination is apt to be interfered with simply from the fall of blood-pressure, and in such patients you have to make up the volume of fluid in the circulation as quickly as possible. The best way of doing this is to give a subcutaneous injection of about 4 ounces of sterilized normal salt solution under the skin of the flank.* This can be injected by means of a clean brass syringe connected with a large hypodermic needle by a piece of rubber tubing. It is very quickly absorbed, raises the blood-pressure, and helps to wash out poisons through the kidneys. The injection may be repeated every six hours if necessary. Now, you cannot, of course, wash out the small intestine, where the micro-organisms are chiefly growing. In order to promote elimination there you require to have recourse to drugs. In other words, paradoxical though it may seem, you must treat diarrhœa, in its early stages at least, by purgatives, just as you treat dysentery in grown-up persons by aperient drugs such as sulphates. You may start with a full dose of castor-oil, which may be poured down the tube after the stomach has been washed out. If there is vomiting and difficulty in getting the oil retained, give half a grain or so of calomel.

There are still some **special indications** to be met. If the skin is in the inelastic state I have described, you will find it advantageous to give the child a wet pack, and you will find benefit also from the administration of diuretics, especially sweet spirits of nitre. If the signs of collapse are great, you may require to have recourse to the mustard bath, the temperature of which is gradually raised to 110° F., this being perhaps the most powerful stimulant we possess. I like also to administer camphor in such cases, either in the form of a solution in olive oil (1 in 15 or 1 in 30), of which 5 minims is injected subcutaneously, or in the form of spirit of camphor, 5 to 10 drops by the mouth. Liquor strychninæ in $\frac{1}{2}$-minim doses subcutaneously is also a valuable stimulant. It may be repeated every four hours. Brandy may also be given in 10-minim doses, added to the albumin water, and repeated as often as is thought necessary.

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* 'Sea-Water Plasma,' which has recently been advocated, is not in any respect better than normal saline.

During all this time the child must be kept under the best hygienic conditions possible. He should be placed in a well-ventilated room, and should be **kept very clean,** the napkins being changed as soon as they are soiled, and never allowed to stay in the room after being taken off the child. In every case the patient should be gently bathed with tepid water twice a day, and the mouth frequently swabbed out with cold water. In bad cases and in very hot weather the child should, if possible, be removed from the town to the country.

I said you were not to put your faith in drugs in the treatment of infantile diarrhœa. But, of course, drugs can give you some help, and I now want to say a few words as to the **use and scope of drugs** in the treatment of such cases.

Although, as we have seen, intestinal putrefaction is an important factor in the production of diarrhœa, I do not think you will get much benefit from the so-called intestinal antiseptics. I am no believer myself in salol and all the substances of that class which are supposed to prevent intestinal putrefaction. I believe the best intestinal disinfectant is a dose of calomel, though whether it acts by virtue of its being calomel or because it is an aperient I do not know.

You may derive certain indications for the use of particular drugs from the character of the stools. If the motions are very sour, it is well to administer alkaline remedies, carbonate of bismuth or aromatic chalk being amongst the best. If, on the other hand, the stools are very slimy and alkaline, many believe in the administration of hydrochloric acid. In the severe cases, so long as the stools are green, I recommend you to give small doses of castor oil in some such form as this :

Ol. ric.	♏v.
Mucilag. tragacanth.	q.s.
Aq. menth. pip.	ad ʒi.

Give this at intervals of two or three hours for one or two days. Instead of castor oil you may give calomel and Dover's powder, ⅛ grain of each. Later on, when the stools have begun to improve, you should give bismuth, remembering the rule to give it in large doses. A very favourite prescription, and one which is now in the hospital pharmacopœia, is carbonate of bismuth, 10 grains ; calomel, ⅙ grain ; pulv. ipec. co., ⅙ grain. Such a powder suspended in some of the albumin water, and given every four or six hours, as the case may require according to its severity, will often be of great service to you after the acuter stage of the illness has passed off. If the stools continue to be watery and offensive, you may require to have recourse to astringents of some sort. Tannigen and tannalbin are very useful in such cases. You will also find in nitrate of silver a very efficient aid—⅛ to ⅙ grain of nitrate of silver is usually sufficient for a young infant. It may be given with dilute nitric acid, 1 minim ; glycerine, 5 minims ; and distilled water, to 2 drachms. Such a dose, given three or four times in the day when the motions are very watery and offensive in the later phases of the illness, is often very useful.

As **opium** plays such a large part in the treatment of diarrhœa, I want to lay down for you, as clearly as I can, some rules for its administration. You can do great harm with opium ; you can kill children with it quite easily. The warnings of the text-books in this matter are fully justified. I have known a baby rendered dangerously comatose by 1 drop of laudanum. On the other hand, there is no one drug which is capable of rendering you such good service if properly used, and in many cases of infantile diarrhœa it is indispensable. Let me state, therefore, the following *rules for the administration of opium*, which I think you will find useful in practice. First, never give it at the outset of the illness. Begin with aperients, not with anything which will cause the contents of the bowel to be retained, as opium does. Secondly, never give it if there are signs of collapse, because then its narcotizing effect becomes extremely dangerous. Thirdly, never give it if the tongue be furred. These rules, like all rules in medicine, are subject to certain exceptions ; but when you are beginning practice let me advise you, if you want to avoid making serious mistakes, to keep them in mind. You will now require some rules as to when you *are* to give opium. Always give it if the stools are very frequent and accompanied by much straining—that is to say, cases in which the lower part of the large bowel is particularly involved are those in which opium is specially indicated. Secondly, if the stools are offensive and the tongue clean, you have a combination of things which cries out for opium, because, as we have seen, this is due to the hurrying on of the contents of the small intestine by too rapid peristalsis. In such a case opium calms down the peristalsis, and so prevents putrefaction and diarrhœa. Thirdly, you should always give opium in those cases in which the diarrhœa is of the lienteric type—that is to say, in which a motion tends to occur immediately after food is introduced into the stomach. Diarrhœa of this type is due to an exaggerated excitability of the whole intestinal tract, and opium is the only agent which can be depended upon to lessen this excitability.

Two practical rules remain. One of these is, never wake a child up to give a dose of opium. If you observe that rule, you are not likely to produce poisoning. The child will sleep off an overdose of opium if you will let him ; the danger is that the mother or nurse may wake him up to give another dose when he is already in a drowsy condition. The remaining rule is that, as far as possible, you should avoid adding opium to other mixtures, but give it by itself—that is to say, measure out a drop or half a drop of the tincture (for the liquid preparations are the most convenient), and give it separately. The reason for this is that one often wants to withhold opium while going on with the other remedies.

The **treatment of chronic diarrhœa** I need hardly describe, because it is conducted on the same general principles as that of an acute case. You do not require to starve such patients, but you select a diet in which there is no milk curd, or in which the milk has been thoroughly peptonized, prevent chill, and choose one of the drugs I have mentioned, according to the indications furnished by the stools.

There remains a special form of diarrhœa which I have not yet mentioned—namely, **ileo-colitis.** I speak of it separately because it is different from the other forms I have described in so far as it tends to be accompanied by ulceration, and to be confined to the large bowel and the lower end of the ileum. It may start as ileo-colitis, or any diarrhœa such as I have been describing may end by passing into a condition of colitis and ulceration. One reason why this disease is of importance is that it is apt to be mistaken for another important disease—namely, intussusception. You will be apt to diagnose colitis as intussusception, and *vice versa*, and that is a mistake which is fraught with serious consequences. For this reason I bring forward the consideration of ileo-colitis separately. How shall you recognize it? Its chief features are that it is accompanied by fever, by straining or tenesmus, and by the passage of blood and mucus. How shall you tell it from intussusception [illegible] intussusception there is less tendency to fever, there is usually vomiting, and you can generally feel the tumour either through the abdominal wall or *per rectum*.* The late Mr. Barnard pointed out a diagnostic sign of some value in distinguishing these two conditions. It depends on the fact that the contents of the small intestine are unable to pass the seat of obstruction in intussusception so that if you get any bile on the napkins, or on the finger after it has been passed into the rectum, you may conclude with fair certainty that you are dealing with a case of colitis, and not of intussusception. If your minds are open to the possibility of making this mistake you are not likely to fall into it often, but I have more than once known the abdomen opened in colitis under the impression that it was a case of intussusception, and I have known one case of intussusception which was allowed to die under the idea that it was one of colitis. So there is a real danger of the mistake being made.

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* In making a rectal examination in a case of intussusception the sphincter will often be found to be relaxed. The descending part of the bowel has been said to feel like the os uteri, but sometimes it is rather soft, and may cling to one side of the rectum, so that the examining finger may pass by it if care be not taken.

About the **treatment of colitis** there is little to say. I have practically described it in speaking of the treatment of diarrhœa in general. As the motions are frequent and accompanied by tenesmus, opium should be given almost invariably, combined with castor oil at first, and later with large doses of bismuth. In this form of diarrhœa you will find washing out of the bowel particularly useful, seeing that the colon is the main seat of the affection. Wash out with warm saline. Some recommend a solution of nitrate of silver, but I think it is of doubtful advantage, and is apt to produce pain. In chronic cases, however, a solution of argyrol or protargol (½ per cent.) is often very effective.

LECTURE VII

CŒLIAC DISEASE

GENTLEMEN,—The question was put to me the other day by one of my colleagues: 'What on earth *is* cœliac disease?' And that is a question which I dare say may have arisen in the minds of some of you when you saw the subject of this lecture announced. Cœliac disease is, as a matter of fact, by no means an uncommon condition, and it is one which is very well defined, although it has not found its way, curiously enough, into the text-books. You may describe it briefly as a wasting disease beginning in childhood, which is characterized by a particular form of diarrhœa, which runs an extremely protracted chronic course with a great tendency to relapses, and which, when it lasts long enough, is apt to lead to a form of infantilism. I shall make it clear as I go along what some of the points in this definition mean.

The disease was first described by Dr. Gee in 1888. He gave a very clear and graphic description of it in St. Bartholomew's Hospital Reports for that year. A few years afterwards it was again described under the term 'acholia' by the late Dr. Cheadle. He used the word 'acholia' because he thought that in those cases there was a deficient secretion of bile. Still later it was re-discovered, so to speak, by the late Professor Herter, of America, who described these cases as examples of 'intestinal infantilism.' He believed they were due to a specific type of micro-organism. These are historical points that do not interest you much, but I bring them forward to show that the disease has been described several times by different people under different names, although it has never found general recognition. And yet it is a disease you will find not so very uncommon in practice. Indeed, it makes up one of those conditions which are slumped together by mothers, in the out-patient department, under the title of 'consumptive bowels,' a so-called disease of which you must all have heard mothers speak.

Passing to **clinical characters,** I have already told you that it is a disease of childhood. It begins not in infancy, but very constantly and definitely about the second or third year of life. The vast majority of the cases, I think, begin in the second year. The first thing noticed wrong with the child, usually, is that it is wasting and is dull, languid and fretful—nothing very definite, but just the general signs of ill-health. If you make inquiry into the character of the stools, you will probably be told that they are rather more frequent than they ought to be; that is to say, not a violent diarrhœa, but perhaps there are two or three or four motions a day. Further, the stools are greatly increased in bulk, and they are usually of the consistence of porridge and not unlike it in colour. They are pale, extremely offensive, and sometimes contain a little mucus or blood.

When you come to examine the child you will find that the only symptom, in addition to the general wasting, is some distension of the abdomen. The abdomen is prominent and tympanitic, but you will not find any lumps, nor any enlarged glands nor fluid. These children, especially after the disease has lasted some time, come to exhibit quite a characteristic mental condition. They are usually rather spoilt children, and as the disease goes [illegible] they get to be remarkably precocious for their age. They acquire what is very unchildlike—an abnormal interest [illegible] their own disease and its symptoms, and may become [illegible] quite hypochondriacal. I remember a well-[illegible] example of the condition in the children's ward here [illegible] a year or so ago, in the person of a little boy three years of age. The sister in the ward said he was only a child [illegible] three, but he talked like a man of thirty—he was so precocious in his ideas and showed such abnormal interest in his own symptoms. This mental attitude has been noticed [illegible] several observers who have written about the disease. The parents also, I might say, tend to be of a certain mental type which may be described as 'fussy.' It might be supposed that this disease is one which affects neglected children chiefly, or children of the hospital class of patients. Quite the reverse is the case. You see some of the best marked examples of it in children who have had a large amount of attention, sometimes, one might almost say, an excessive amount of attention paid to them from their infancy upwards, and who have been brought up amid the best surroundings.

As the condition progresses certain **complications** may appear, the cause of which we will consider later on. Amongst these is weakness of the legs. This may set in fairly early. I have known a case in which difficulty of walking was one of the first things the mother noticed wrong. In all cases, if they have gone on for some time, the child tends to get weakness of the legs accompanied by absence of knee-jerks—a condition which looks like one of peripheral neuritis. They may get also œdema especially affecting the arms, hands, legs and feet, without any renal disease — one of those 'toxic' or 'essential' œdemas, as they are sometimes called, which you are apt to meet with in young children who have suffered from chronic intestinal disorder for some time. That you will see in a fair number of cases.

A much more severe complication is tetany. There may be tetanic spasm of the hands and feet, and you get also in a few cases actual general convulsions, epileptiform convulsions.

These, then, are the clinical characteristics: wasting, diarrhœa characterized by stools that are not numerous but large, pale and offensive; distension of the abdomen; a curious mental attitude; and certain complications such as weakness of the legs with absence of knee-jerks, toxic or essential œdema, tetany and sometimes convulsions.

The disease is a very chronic one, it runs a long course which can be measured by years. It has also this aggravating peculiarity, that it exhibits a great tendency to relapse. Just when everything seems to be going well and improvement setting in, suddenly there is a set-back and all the symptoms become aggravated much more. This is quite a well-marked feature of it. You must not suppose, however, that it is necessarily a fatal disease—very far from it. It may be completely recovered from even if it has lasted two or three years, and the child become perfectly sound and well to all appearance, but, of course, you may have death in the course of the disease, usually from some intercurrent trouble such as broncho-pneumonia.

There is a group of these patients which is very interesting—those, namely, where the disease has lasted a long time, and has interfered so much with growth and development that **infantilism** results. This tendency for the disease to interfere with growth and development was well recognized even by those who first described it, but it is only lately that the late Professor Herter wrote a little book on "Intestinal Infantilism" in which great stress is laid on the arrested physical development. I think, myself, there is no great mystery about the infantilism. It is probably simply the result of chronic starvation. Any disease which interferes with nutrition will finally lead to a condition of retarded growth, and any condition of retarded growth and development one may speak of as infantilism. The child, as regards its physical condition, appears two or three years younger than it really is.

As regards **diagnosis,** the only thing that cœliac disease simulates is abdominal tuberculosis. The distinction between the two is made all the more difficult by the fact that cases which begin as cœliac disease or intestinal cararrh are apt to become secondarily infected by tubercle and pass on abdominal tuberculosis, and it may be difficult to say when this has taken place. But you are never justified in diagnosing tubercle unless you can feel enlarged glands in the abdomen, or a thickened omentum, or make out the presence of free fluid. The tuberculin reaction will help you in some instances. A negative reaction, I think, will justify you in the case of a young child in excluding tubercle.

The **pathology** of cœliac disease is still disputed. One thing everybody is agreed upon is that if you make a post-mortem examination in one of these cases you will not find anything beyond the fact that the intestinal wall is rather thin and atrophied-looking, but there is nothing to be made out on careful inspection of all the organs. I have had a post-mortem examination on two cases, and in neither of them could I find anything visibly wrong at all. In the absence of a definite naked-eye pathology, various theories have been put forward as to the cause of the symptoms. It has been suggested, for instance, that they are due to functional deficiency on the part of the liver; that the liver does not secrete enough bile, or that it does not perform its duty adequately. This was the theory of Cheadle. A second theory is that the pancreas is not sufficiently active. A third theory is that the condition is simply due to catarrh of the intestine, possibly set up by a specific type of micro-organism. That is the theory which Professor Herter adopted.

In deciding between these theories, careful examination of the stools might be expected to be of help. If you examine chemically one of the large pale offensive motions, in a case of cœliac disease, you will find always a great increase in the amount of fat. About that there is no doubt. The pale colour of the stools is mainly due to insufficiently absorbed fat. I have never seen a case where the bile pigment was entirely absent from the stools; with the perchloride test you can get a bile pigment reaction. There is also no other evidence of inactivity of the liver in these cases. There is no defective secretion of urea, for instance, so that one can only say there is insufficient evidence in support of the view that the liver is inactive. It may not be as active as it ought to be, but there is certainly no complete arrest of the bile-secreting function. In favour of the pancreatic theory is the imperfect absorption of fat. But you will find that most of the fat that occurs in the stools in these cases is fat already split up into fatty acids and soaps. In cases where the pancreas is inactive, the form in which the fat is met with in the stools is ordinarily fat unsplit up. But still, in some cases of the disease there is a great deal of unsplit-up fat present, looking as if there were perhaps a deficiency of pancreatic secretion. In favour of that view, also, is the fact that these patients cannot stand starch. If you give them much starchy food, you will find unaltered starch in the stools, so that there is a certain amount of evidence in favour of the pancreatic view. To that one might add that, undoubtedly, some patients are much benefited by giving them active pancreatic ferments by the mouth.

There is, finally, the view that the whole clinical picture is simply the result of intestinal catarrh. In favour of that is the fact that many of these cases begin with acute intestinal catarrh. A child, for instance, gets an ordinary acute attack of diarrhœa from which it does not make a complete recovery, but goes on by degrees to exhibit the full-blown picture of cœliac disease. Then, of course, the intestinal catarrh would explain the character of the stools ; because, if the contents of the intestine are hurried on too fast, there is no time for the absorption of fat, so that a large excess of fat in the stools is quite compatible with the view that one is dealing with an intestinal catarrh, and the very offensive character of the motions also may result from that : the contents of the small intestine being hurried on rapidly into the large intestine, where they putrefy. So that on the basis of intestinal catarrh you can explain all the characters of the stools fairly well. Professor Herter went further, and adopted the view that the catarrh was set up by a specific type of micro-organisms. He pointed out that in the stools in these cases you get a great excess of gram-positive organisms. In the normal stools you get an excess of gram-negative ones. Dr. Panton examined for me the stools from two cases of cœliac disease, and there was no doubt about the positive organisms being relatively in excess, but he was unable to find specific types of abnormal organisms such as Herter believed actually caused the disease. I think it is an open question whether the organisms present in these cases are not the result of the state of the intestine rather than its cause. Abnormal flora you undoubtedly get, but I think they are quite as likely to be consequences of the catarrh as the cause of it. Whether the condition is due to specific organisms or not, I think that all the evidence is in favour of the view that these cases are to be reckoned essentially as cases of chronic intestinal catarrh, where absorption of the food constituents is greatly interfered with, and possibly there is in some of them some degree of defective action on the part of the pancreas as well.

We now come to the important question of **treatment.** The first and most important thing in the treatment is to find a suitable diet. Seeing that fat is so badly absorbed in these cases, one must greatly lessen the amount of fat in the food. I told you also that most of these children do not stand starch well. If you give them starchy food, you will find unaltered starch in the motions, and with that you get fermentation and great abdominal distension, so that you have also to limit the amount of starch or give a substitute for the starch in the form of dextrinized foods. Proteins the patient can stand quite well, and gelatin is also a particularly useful food constituent, so that it comes to this : the constituents of the diet must be proteins, gelatin, and dextrins. It is not quite easy to construct a diet on these lines, and yet successful treatment depends entirely upon your doing so.

I should recommend that you always make raw underdone meat the basis of the diet. Children two years old, or even less, will take six ounces of raw meat daily quite well. It is made by scraping down the meat, so as to remove the fibre from the connective tissue, and may be given with a little broth. The only objection to it is the possibility of producing tape-worm. That happened in one patient that I had, but it is rare, and in any case it is easy enough to deal with the tape-worm afterwards ; but there is no question at all about the immense value in these cases of raw meat. In addition to the raw meat itself, you can also give with advantage pure raw-meat juice. I do not mean the bottle juices, I mean the actual juice of the meat squeezed out by means of a press. You can buy now a small press (the Hercules Meat Juice Press), and by turning a screw you can squeeze from the meat all the juice part, which looks just like blood, and which forms a liquid of high nutritive value. Several ounces of this can be given in the course of a day. The raw meat and meat juice should be, so to speak, the *pièce de résistance* of the diet. Dextrins you can get in the form of Mellin's food biscuits, grape-nuts, breakfast biscuits, and malted rusks, which contain little or no starch, and can be given freely. Herter is a great believer in gelatin; he thinks it does not form a basis for the nourishment of the particular organism which he believes is at work in these cases. Certainly children stand it well, and it is nourishing. You can give it in the form of calves'-foot jelly or any form of food into which gelatin can be introduced. Milk, these patients do not stand well, partly because it contains fat and partly because the curd of the milk is not easily digested; but you can give skimmed or peptonized milk, and, used cautiously, you will find it of value. Fortunately, there is not usually much difficulty in getting the child to take this diet. The improvement in the stools under it is immediate.

Next in importance is the hygienic treatment, and one of the chief things to attend to here is the avoidance of chill. These children have a great tendency to get cold extremities, and even the least exposure to cold may bring on a relapse and a return of the diarrhœa. Hence you have to guard them very particularly against chill. The legs have to be kept very warm; sometimes one may have to wrap them in cotton-wool and bandage them; these patients should also wear a flannel binder in order to keep the abdomen warm. Be careful also that they do not get chills out of doors or when they are being bathed. Further, you will often find a great advantage from change of air. If a case is not making progress, a change of air to the seaside may help matters very greatly. In a protracted sort of illness like this, it may be necessary several times in the year to send the child away for a change.

Last we come to the treatment by medicines. Medicines help you a good deal in the treatment, but they are secondary in importance to the diet and general management. First I would put opium. It is particularly serviceable: it lessens the motions by delaying the passage of food through the small intestine, so allowing of better absorption, and, by preventing the food being hurried on into the large intestine before it has been absorbed, it lessens putrefaction, because by the time the contents of the small intestine reach the colon there is less unabsorbed material to putrefy. Hence, under the use of opium the stools become less frequent and less copious and less offensive ; the nutrition also tends to improve as the child gets more value out of its diet. You may give 1 minim of tincture of opium three times a day, for every year of the child's age, with a carminative mixture or with an astringent such as bismuth, or with a more powerful astringent such as nitrate of silver, which I have often used with great advantage. One-sixth of a grain of nitrate of silver may be given along with a drop or two of dilute nitric acid and a little glycerin and distilled water. Some people are afraid of using silver in case of producing pigmentation, but I have never known that happen. By omitting to give it for a week or two now and then you can obviate any such danger.

Under the heading of medicines one may consider the question of ferments. Where there is much unsplit-up fat and unaltered starch in the stools it is well worth while to try an active preparation of pancreas. One of the best of these preparations for children is that known as 'Pankreon,' given in the form of sugar-tablets, two after a meal. Another good preparation is the American one called 'Holadin,' which may be given in capsules, one three times a day, two hours after a meal.

LECTURE VIII

CHRONIC CONSTIPATION IN INFANCY AND CHILDHOOD

GENTLEMEN,—Of the minor maladies of early life there is none commoner, or more far-reaching in its consequences, than that which I have chosen for my lecture to-day—namely, chronic constipation. And the subject is of interest, not only on account of its immediate significance, but because it is very probable that the habitual constipation of adult life, which everyone knows is one of the most prevalent of the minor maladies of the people at large, often owes its origin to the neglect of habitual constipation in childhood. For it must be remembered that the bowel, after all, is a creature of habit, and if it is to be trained up in the way it should go its education must be begun early, and carried out persistently. I feel perfectly certain that if habitual constipation in early life were taken more seriously, and were more perseveringly treated, we should hear much less of chronic constipation in older persons than we do.

It will be convenient for our purposes this afternoon to consider, first, chronic constipation in infancy, and, secondly, chronic constipation in older children, because one meets with the disorder both in the early years of life and also in what one may speak of broadly as later childhood.

CHRONIC CONSTIPATION IN INFANCY.

I think one may say at the outset that constipation in infancy is much commoner in breast-fed than in bottle-fed children. I know that in some text-books the very reverse is stated, but I believe that to be a mistake, and I think anyone who keeps his eyes open here in the out-patient room will have seen that when an infant is brought for constipation it is almost invariably a breast-fed child.

It is difficult to say why it is that breast-fed children should be so apt to suffer from chronic constipation ; certainly I have not satisfied myself as to the reason for it. In some cases it may, perhaps, be due to *some peculiarity in the mother's milk*. For instance, one might mention as one possible peculiarity a deficiency of fat in the milk, and that certainly may be a cause in some instances. On the other hand, I feel certain that in some of them it is due not so much to deficiency of any ingredient as to the milk being altogether too poor in quality, too watery, with the natural result that it is absorbed almost completely, so that there is practically no residue left to stimulate peristalsis. And in favour of that view is the fact that in many cases the habitual constipation is accompanied by persistent wasting, showing that the child is getting an insufficient amount of nutriment. That may explain some of the cases, though certainly not all of them.

Then, again, there are cases of constipation which are accompanied by colic, where the child is more or less restless, draws up his legs, and shows all the signs which one associates with spasm in the bowels. And one finds not uncommonly that if one can remove the colic the constipation also disappears. In these cases I think we may reasonably speak of the condition as one of *spasmodic constipation*. There would seem to be a spasmodic contraction of the bowel which is the cause of the colic, and which is at the same time an obstacle to the onward passage of the intestinal contents.

There is another group in which there is reason to suppose that *the intestinal secretions are defective*. For instance, you will meet with cases in which the mother says that the stools are exceedingly hard, friable, and even chalk-like in appearance, and in those cases, I think, one is right in supposing that there is a defective secretion on the part of the intestinal glands, and possibly also defective secretion on the part of the pancreas and the liver, and that it is to this that the constipation is due.

Again, it is possible, although it is difficult to prove it, that in some of these cases one has to deal with *congenital atony*, or want of power in the wall of the bowel. Combined with this congenital muscular feebleness there may also be a deficiency of reflex nervous excitability, but there is no reason that I can tell you why that kind of constipation should be commoner in breast-fed than in bottle-fed babies.

There is another point about constipation in infancy to which I should like to direct your special attention, and it is that, for some reason or another, it is particularly common and obstinate in children who suffer from any form of mental deficiency. The forms of mental deficiency in which it is particularly apt to occur are cretinism, and what one can best term infantilism. Perhaps one should not really speak of infantilism as a form of mental deficiency, but it is at all events an arrested development in which to some extent the brain participates, and in connection with these conditions I have seen some of the most obstinate and severe forms of habitual constipation in infancy which one has ever met with. And the peculiar thing is that in such cases the administration of thyroid to the child may act almost as a laxative. A few years ago I had to treat a baby, the chief complaint being that it suffered from constipation, and always had suffered from it to an extreme degree, so that a week or ten days might elapse without a spontaneous motion. I tried all sorts of aperients, but without success, the efforts extending over a long time. The child was born in South Africa, and the mother had consulted several people in this country about it, as well as on the Continent. The story she gave was that various laxatives had been recommended, but that nothing had yielded success. One day it occurred to me that the child looked slightly cretinoid ; that is to say, although it was certainly not a well-marked cretin, still, there were features which suggested cretinism. So I started to give thyroid with the laxatives which had been prescribed, and practically from the moment that combination was given the difficulty with the bowels disappeared. That is a good example of the fact to which I wish to direct your attention —namely, that habitual constipation is sometimes associated with mental deficiency, and in such cases I believe it to be due to impaired innervation. My opinion is that in these children the complicated nervous mechanism controlling peristalsis is either imperfectly developed, or at all events sluggish, just as one knows that other nervous mechanisms are interfered with—for instance, that which controls speech, or that which controls the complicated muscular movements concerned in the act of walking. These children are late in learning to talk and to walk, and I believe that they are also late in developing a proper peristaltic control.

Of the **consequences of habitual constipation** in the infantile period of life I have mentioned one—namely, *wasting*—and that is perhaps one of the commonest consequences of it, or, at all events, it is one of the commonest concomitants of the sluggish action of the bowels. I have said that in some cases this wasting is to be explained by the poverty of the mother's milk, which not only causes constipation in the way I have indicated, but also causes wasting. But that certainly is not the cause of the wasting in every case, because it not infrequently happens that when one gives an aperient to such a child, and begins to get a regular action of the bowels, it begins to gain weight straight away, even though there is no alteration in the feeding. It would seem, therefore, that there must be some cause for wasting in these cases other than mere deficiency in the constituents of the milk. What that other cause is it would be difficult to say. I think that in some cases it is due to deficient secretion of the intestinal juices of which I spoke, which leads to imperfect digestion of the food, or perhaps to imperfect absorption of it. And when, by suitable aperients, one stimulates the secretion of the intestinal glands, digestion and absorption are both improved, and in consequence the weight begins to go up. I think that in addition there is some reason to suppose that in some cases the wasting is due to the continual restlessness which the constipation causes. The child is kept constantly uncomfortable, and as the attacks of colic come on he screams and kicks about very vigorously. Also he sleeps badly, so that altogether he expends a great deal more muscular energy than does the normal child of that age. This all means a waste of tissue.

Then, there are certain mechanical consequences of habitual constipation which should be borne in mind. One of them is the result of the continual straining which the constipation induces. This strain falls most on the abdominal wall. And one of the most obvious of these results is *the development of hernia,* and particularly umbilical hernia. One often sees umbilical hernia in children who are the subjects of habitual constipation. Another result of this constant straining to pass a motion is the occurrence of a partial *prolapse of the rectum.* There is a third and rarer consequence of habitual constipation which must be mentioned, and that is *dilatation of the colon.* The exact mode of production of this condition, which is also known as 'idiopathic dilatation of the colon,' or 'Hirschsprung's disease,' is still in dispute. I have spoken of it as if it were the result of prolonged constipation, but it is only right to point out that many believe that dilatation and hypertrophy of the colon may occur as a congenital defect, and be accompanied by obstinate constipation as one of its symptoms. Whatever the correct explanation may be, you will occasionally meet with cases—nearly always in boys—in which there is a history of inveterate constipation dating from birth, and in which you find the abdomen to be greatly distended, tympanitic, and often exhibiting visible colonic peristalsis. I had a case of this sort lately in a baby of ten months, in which there had been no action of the bowels for ten days in spite of the vigorous use of enemata. If you examine such a colon after death—and the cases are not infrequently fatal—you will find that it is enormously dilated throughout most of its length, and the wall greatly thickened, just as the wall of the stomach is in cases of pyloric obstruction. The curious thing, however, is that no actual cause of an obstruction can be discovered, certainly none of an anatomical sort. Hence the explanation has been advanced that the obstruction is really a physiological one, and consists in a neuro-muscular defect, in consequence of which a section of the colon is incapable of forwarding its contents.

The treatment of dilatation of the colon is really surgical, and I only mention it here because of its association with constipation.

Finally, there are *reflex consequences* of this habitual constipation, the result of the stagnation of intestinal contents, and the irritation by those contents of the wall of the gut. Amongst those consequence, perhaps the most marked are disturbances of sleep and restlessness, often accompanied by twitching movements, and sometimes culminating in convulsions. On the other hand, little babies seem to suffer much less from the toxic consequences of constipation than do older children. That is probably attributable to the fact that the diet of infants consists almost entirely of milk, and that milk produces a less degree of intestinal putrefaction than does any other article of food.

I shall now speak of the **treatment of habitual constipation** in infancy. And first I would emphasize that the treatment must be *persistent*. You will find that the treatment adopted by the mother in such cases tends to be anything but persistent. The child is in most cases allowed to go on without a motion for two or three days, and not until then is anything done, and then the mother gets alarmed and flies to castor oil or some other drastic aperient. This acts at the time, and then the child is allowed to go on again for another two or three days, or perhaps longer, and the treatment is repeated, and so on. That, of course, is not the way to deal with a case of habitual constipation in an infant. The treatment of these cases should not be by means of drastic aperients, but by means of those aperients which exert a more or less tonic action on the bowels. Your object should be in all these cases to educate the bowel to act spontaneously ; you do not want to have to go on giving aperients always, but to bring about a condition of affairs in which the bowels act spontaneously. Such aperients as aloes and cascara sagrada are the best, and in many cases one has to fall back upon one or other of those in order to produce a suitable action. But before I proceed to speak of the methods of prescribing aloes and cascara, I want to mention one or two other plans which are sometimes sufficient in the milder cases. In bottle-fed

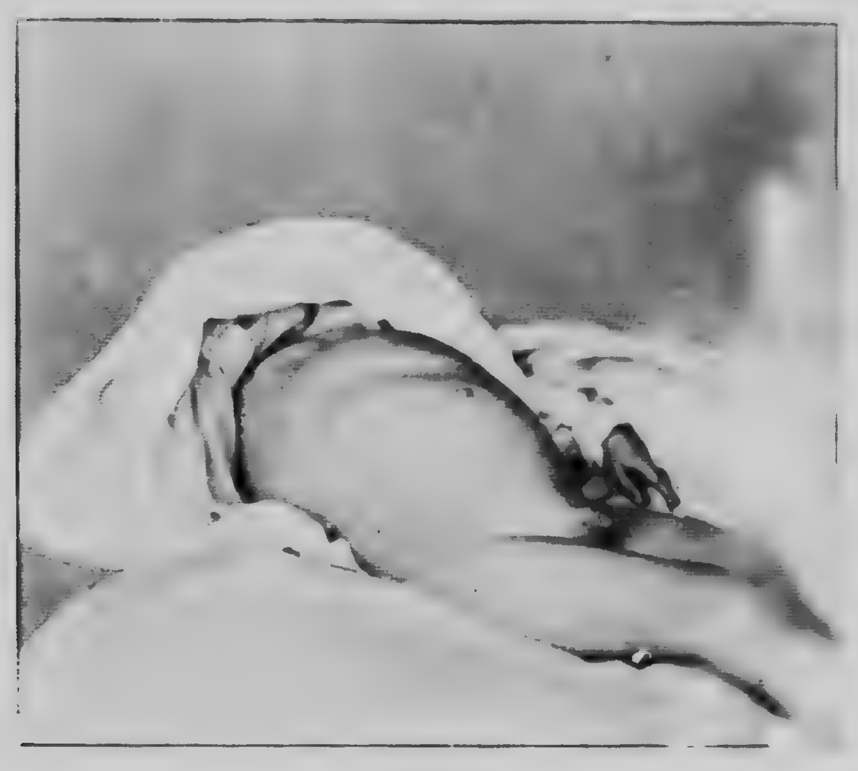

FIG. 5.

CONGENITAL DILATATION OF THE COLON, SHOWING DISTENDED LOOPS OF BOWEL.

babies, for instance, you will find that adding phosphate of soda to the bottle is often enough to produce an action. You should add 5 to 10 grains to each feed. Or one may sometimes give manna, which has a laxative effect. It should be dissolved in hot water and added to the food. Or sometimes a grain or so of sulphur given with the baby's milk produces the desired effect. In breast-fed babies fluid magnesia is often found to be a useful aperient.

On the other hand, there are certain methods of treatment which you ought to avoid. One of these is any attempt to treat constipation in the child by administering aperients to the mother. One can only call that a very roundabout and unsatisfactory method of trying to reach the child's complaint. Many people believe, however, that that method of treatment [illegible]acious, and you will find that sometimes sulphat[illegible]gnesia is administered to the mother with that objec[illegible]t you will think it over you will see that the only chanc[illegible]f this acting is that some of the sulphate of magnesia may be excreted in the milk, and so reach the child's intestine. If the mother suffers from constipation there is no constipating principle excreted in her milk that I know of, and the only conceivable way in which laxatives could act upon the child if given to the mother would be by reaching the child through the milk. But that is so uncertain that it is not a method which could commend itself to any rational therapeutist. Again, I do not think the tendency to administer aperients by the lower bowel is much to be commended. That is a method largely in vogue in the nursery, in the form of soap suppositories or small injections. In some cases that treatment is good enough—that is to say, in those cases where the constipation is due entirely to stagnation in the large bowel and the defective action of the lower part of the large intestine. But if, as I think there is reason to suppose, many of those cases are due to a cause operating much higher up in the intestine, such a method of treatment is not to be commended, because it acts upon one section of the bowel only. The habitual use of laxative enemata or suppositories is also apt to result in a certain amount of irritation of the rectum, setting up a chronic catarrh or proctitis.

For these reasons this method of treatment is not to be commended.

As an accessory to bringing about a proper and regular action of the bowels, *massage* is always a plan to be adopted. It undoubtedly helps where the constipation depends upon a defective muscular tone in the large bowel, and it can easily be carried out by the mother by rubbing the abdomen along the line of the colon systematically for five or ten minutes two or three times a day, at the same time kneading the bowel. If necessary, a small amount of any unguent or oil may be used to prevent breaking of the skin, but I think it is futile to expect that by rubbing anything on the abdomen it will find its way in, and produce laxity of the bowels in that way. One has known an aloetic preparation used with the idea of it being absorbed ; but that, like the method of trying to bring about an action of the bowels of the baby by giving the mother a laxative, is very unsatisfactory. The oily substance which you rub on the abdomen acts mechanically, if it acts at all, and is only used to prevent irritation of the skin, which might otherwise follow the rubbing.

I have said that one has to fall back in all obstinate cases upon one or other of the tonic aperients, such as aloes or cascara. Aloes is best given to little children in the form of the tincture. For a child aged six months 3 to 5 minims will be sufficient. With that you may combine some other aperient drugs, because in children, as is probable also in adults, aperients often act best when given in combination. One form of aperient acts on the muscles, another on the nerves, and another on the secretions, and you want to combine the actions of all. We do not know exactly what is the actual factor at work in the production of any given case of constipation. Certainly I do not pretend to know definitely whether a given case of constipation is due to defective muscular action, or to defective secretion, or to defective nervous control ; therefore in the treatment it is better to give a small amount of an agent which will act upon each of these different constituents. For instance, one can give 3 or 4 minims tincture of aloes with the idea of stimulating the peristalsis of the intestines, and also

10 grains of sulphate of soda to increase secretion. One has to remember also that aloes has a griping action, and it is necessary to put something into the medicine to prevent it. Of the preparations with that action, one of the best is belladonna, so that one adds about 1 minim of the tincture of belladonna to the mixture. It is always well to give in addition a carminative (such as syrup of ginger, 20 minims), and peppermint-water to 1 drachm. Some such mixture as that may be given both night and morning. or it may be given three times a day, or oftener. One has to find out what dose is required to produce a daily action in the case with which one has to deal, and, having found what dose is required, one should maintain it for a number of weeks. It should then be gradually discontinued, and probably one will find that by the end of that time the bowels have learnt to act spontaneously. Sometimes even that combination of aperients is not sufficient. Some of these cases are so obstinate that even stronger measures are necessary to bring about what is desired. And in such a case you may add syrup of senna, which acts on the small bowel as well as the large one, 15 to 20 minims of the syrup being added to each dose of the mixture. I have recently used in some cases of chronic constipation in infancy the new synthetic aperient which goes by the name of 'purgen.' It produces fairly satisfactory results if given in doses of ¾ to 1 grain twice a day, but I think it acts mainly by stimulating secretion, and does not seem to exercise any tonic effect, such as does aloes. It has the advantage of not griping.

Some special indications for the use of drugs may be gathered from the study of the motions. If they are white, chalky, or friable, you will find that there is nothing so good as podophyllin. It is best given in the form of the tincture, 1 or 2 minims two or three times a day being the dose. It may either be added to the mixture which I have described, or it may be given by itself. If there is much straining, and the motions are hard and passed with much difficulty and tenesmus, I think you will find that sulphur gives specially good results. I use it in the form of the confection, in half-teaspoonful doses. It softens the motions in a wonderful way, and children take it well.

CONSTIPATION IN LATER CHILDHOOD.

So much for constipation in the period of infancy. Now we pass to consider its occurrence in later childhood. Here, as I indicated at the outset, the malady is often the result of the neglect of constipation during infancy, but in some cases that is not the cause, the constipation being acquired, and coming on after the child has cut the second teeth. In some of these cases it is due to defective diet, to the child not getting a sufficiency of those things which stimulate peristalsis. That is to say, the diet is either altogether deficient in bulk, or it is deficient in those articles which contain a quantity of cellulose which one knows are specially calculated to produce an action of the bowels. I think it is easily possible to exaggerate the importance of the dietetic causes of constipation, and as an inference from that we may safely say it is easy to exaggerate the amount of improvement which you are likely to obtain by an alteration of the diet. I am certain of this, that there is scarcely any case of constipation bad enough to be brought to the doctor for treatment which can be treated effectively by change of diet alone. It sounds plausible that if you give things which will stimulate the bowels, bulky things or those which contain stimulating principles, you will effectually remedy the condition. But I am perfectly certain that if you attempt to carry that out in practice you will be disappointed in the majority of cases. Further, if your dieting fails to do good, then you may be certain you will end by doing harm; for if you increase the bulk of the diet without remedying the constipation, the result is that the bowel has still more work thrown upon it, the accumulation becomes greater, and the chances of dilatation and atony are increased. So I would say to you that if your dietetic means do not answer at once they are likely to do more harm than good if persisted in. After all, there is nothing very mysterious in the way in which dietetic means act. They either act mechanically by stimulating peristalsis, or they act—as prunes, for instance, do—because they contain laxative principles, and it is much easier to regulate a dose of senna than a dose of prunes.

In the one case you know exactly what you are doing, but in the other you do not, and I do not see why prunes should be preferred as an aperient to senna. It is true one is a common article of diet and the other is not, but that is not a special commendation. So, although a modification of the diet may be necessary in such cases or advisable, in all your more severe cases you will require to fall back on drugs.

In other cases of habitual constipation in the later period of childhood the condition is due, no doubt, as in the adult, to some secretory or muscular fault in the bowel, or to some sluggishness of reflex action, or possibly to a combination of these causes—that is to say, to deficient innervation along with defective muscular action and secretion. And, as I have said, I do not think anyone can pretend to know with certainty which of these causes it is that is at work in any given case. I would warn you here about a fallacy into which one is tempted to be led, and that is of mistaking an insufficient action of the bowel for a natural action. Again and again you will be told by the mother that the action of the child's bowels is regular. And so it probably is, but it is insufficient ; there is not a complete evacuation every day ; only a certain amount is passed, while a certain amount is retained. The inference from this is that if you are dealing with a child whom you believe from its symptoms to be subject to constipation, you should not be deterred from administering aperients just because you are told by the mother or the nurse that the bowels act regularly. Over and over again I have found great benefit from giving aperients to children in spite of the assurance that the bowels were regular.

The **results of constipation in later childhood** are even more far-reaching than in infancy, and for this reason : that you have the toxic effects superadded to those which are due to reflex irritation, which I have mentioned as being chiefly at work in the case of the infant. You will find that older children who suffer from constipation are always dull and languid ; they suffer from headache and defective appetite, and in particular from disturbed sleep. They are restless and nervous at night. Again, you will find that, as a consequence of the toxæmia, their tempers become affected, and they are depressed, irritable, and peevish. You will also find, as a result of the retention of the intestinal contents and the production of an excess of mucus by the irritation of the scybala in the bowel, that the lodgment of worms in the intestine is encouraged; indeed, I would say that the treatment of threadworms in most cases consists in giving regular laxatives. You will find there is a rarer consequence of constipation, and one which is apt to be overlooked, and that is *intermittent pyrexia*. You will have cases brought to you every now and then—and very puzzling they are unless you are warned about them—in which the mother tells you that the child at intervals of a few weeks becomes feverish. There is nothing else obviously wrong with the child, but at night the temperature goes up to 102° or 103°, and that sort of thing may go on for several nights in succession, and may subside without there being any other special manifestation of illness. I saw not long ago two little girls, in both of whom the history was that the child had suffered for some months from the attacks of pyrexia such as I have mentioned. All sorts of treatment had been adopted, but without any benefit. I was told that the bowels acted regularly, but, in spite of that, I gave aperients, and pushed them until there were two liquid actions every day. That caused the pyrexia to disappear. How the pyrexia is brought about in such cases I do not know. It may be toxic, but, at all events, I would say that if you have a child brought before you which suffers from recurrent attacks of pyrexia without any apparent reason, you will do wisely to order that child aperients, and in the majority of cases that treatment alone will be successful.

Now to pass to the question of the **treatment of constipation in later childhood.** You will give your attention, or direct the mother's attention to the ordinary hygienic rules, to the importance of suitable diet, if necessary, to regular exercises, to massage of the abdomen, or perhaps abdominal exercises, and sometimes even to cold douching or sponging of the abdomen. All of these measures help, and should be tried first. In some cases, again, you will find it advantageous to order larger quantities of liquid to be drunk. There are some children who do not drink enough water, and in certain cases, where the motions are dry, you will find that merely drinking larger quantities of fluid is sufficient to bring about a satisfactory result. But in the majority of cases you will find that these means alone are not sufficient, and at last you have to fall back on the use of aperients. Here the principle which I laid down for infants applies with greater force ; that is to say, the treatment which you adopt must be persistent and continuous, and not spasmodic and intermittent, and the drugs which are used should be those which have a tonic effect on the whole of the bowel. Here, again, cascara is very useful, given preferably as the elixir, or you may give aloes in the form I have indicated. Of course, you will require to increase the dose in accordance with the age of the child. In some cases quite a large dose will have to be given. I have had to give as much as a drachm of sulphate of soda and 15 minims of tincture of aloes, with some syrup of senna, three or four times in the day before a satisfactory result was produced. You should give an increasing dose until you get at least two liquid motions in the day. Having reached that point, you continue the same dose for some weeks, and then gradually leave it off, for at the end of that time you hope to have brought about a spontaneous action and educated the bowel to perform its function properly. Here, too, as in the case of little babies, there are sometimes special indications to be derived from the character of the symptoms. Where, for instance, there is reason to suppose that there exists a sluggish action of the liver—whatever that may mean—where there is depression of spirits, and a yellow conjunctiva, and a furred tongue, you only get the best results by giving a so-called hepatic aperient in addition to the other drugs which I have recommended, and one of the best of these is mercury. One may give it in the form of gray powder at night in addition to the aperients I have described. If there is much mucus in the motions and worms are present, I prefer to give mercury along with rhubarb, 5 to 10 grains of powdered rhubarb, with 1 or more grains of gray powder, being given in the form of a cachet. You will find that the rhubarb has a tonic effect upon the mucous membrane—or an astringent effect, as some regard it—and one of the consequences of its administration is that mucus ceases to be secreted in such a large amount as before. The result is that the worms no longer find lodgment, and so they disappear. But rhubarb has not that tonic effect on the muscular part of the bowel which belongs to aloes and cascara. It has an astringent after-effect, for which reason many people object to its continuous use. But in many cases, in combination with gray powder, it gives better results than anything else. That is the principle to go upon in your treatment—to select a tonic aperient and administer it persistently. If you are persistent, it does not matter very much what laxative you give; you may find one child will react better to one form, and another child will do better on another form. The great thing to bear in mind is the education of the bowel, and in a child it should always be possible to do that. In chronic constipation in the adult the bowel seems to be almost ineducable, but in the case of the child one ought always to be able, by persistence, to train it properly. By persistence one means treatment carried out often over weeks, or even months, and it is always well to warn the parents that the treatment must necessarily be prolonged, and that they must not be discouraged. If this persistence is practised, I am sure that the reward will be, not only the relief of the immediate symptoms, but the prevention, possibly, in after-life of habitual sluggishness of the bowels and all the consequences which follow in its train.

LECTURE IX

THE WASTING DISEASES OF INFANCY—MARASMUS AND CONGENITAL SYPHILIS

WASTING.

GENTLEMEN —In this lecture I wish to consider with you *wasting* as a symptom of disease in infancy. When you go into practice it will occur to you almost every day to have a baby brought to you with the complaint that it is wasting.

The mother will tell you, in the common phrase, that it was 'a beautiful baby born,' but that either from birth or at some period subsequent to birth it began to waste. And very commonly you will find on inquiry that the child began to waste when artificial feeding was adopted. I would point out to you that wasting is not a disease in itself ; it is, strictly speaking, a symptom, not a disease. So when a child is brought to you with this complaint it is your duty to try to find out to what the wasting in that particular case is due ; and there are many possibilities which should present themselves to your mind.

The first is that **the feeding may be insufficient.** That is the first possible cause of wasting, and, of course, a most natural one. If the case be one of a breast-fed child, you may find on analysis of the milk that it is poor in fat, or that the total quantity secreted is small. If it be a bottle-fed child, you will find that by increasing the strength of the mixture on which the child is fed, or by giving more at each feed, it will start to gain weight. So there is no great difficulty in diagnosing wasting from mere insufficiency of nourishment.

Then you may have, as a second cause of wasting, **inability on the part of the child to suck.** That is a cause which you are more likely to overlook. The child may be unable to suck the breast or the bottle because of local conditions—I mean such things as stomatitis, an inflamed and painful condition of the mouth, which makes the movements of the tongue in sucking productive of acute pain. In such a case the child will cease to suck, and whilst the local condition lasts he will waste. But that, as you will see, is not likely to be a cause of chronic or prolonged wasting, because those local conditions in the mouth are, fortunately, usually of only short duration. Then, a child may be unable to suck because he is unable to breathe. I mean that if the nose be blocked up the child has only one channel left by which both to breathe and to feed, and in that case he is unable to breathe whilst sucking; and owing to that he is probably unable to get down a sufficiency of nourishment, and so again wasting occurs. Other local causes are such conditions as hare-lip or cleft-palate. I have known children waste severely on account of their inability to suck from the presence of a cleft-palate.

In the third place, I would remind you again of the possibility, in the case of bottle-fed children, that **the teat may be unsuitable.** I saw a child not long ago, for instance, which was brought to me when a month old, and weighed only 6 pounds 7 ounces. At first I could find no obvious cause for the wasting. I made a little modification in the feeding, and it was brought a week later weighing 6 pounds 5 ounces. Still I could find no explanation for the loss of weight. The child came back a week later weighing 6 pounds 4 ounces. Then I looked at the teat, and found it was of the 'leech-bite' form, which, as I told you before, often prevents the milk coming through easily. I recommended that the bottle should have a new teat, which was done, and the child was brought back the next week weighing 6 pounds 10 ounces, and in a fortnight it weighed 7 pounds 12 ounces. That is an example of how real this cause of wasting may be, no matter how trivial it may appear.

In the fourth place, an infant may waste on account of

persistent indigestion—that is to say. if a child is getting a mixture which is too strong for him, a mixture which is chemically quite sufficient to supply his nutritive requirements, but which is too strong for the digestive organs. that child will waste, with the ordinary symptoms which I described to you in another lecture—namely. colic. vomiting. and probably occasional diarrhœa. So that the treatment of this form of wasting will resolve itself practically into treatment of the disorders I described to you earlier in the course.

A fifth cause, and one which you must never overlook, is the possibility of **the existence of organic disease.** I refer particularly to congenital syphilis, to latent tuberculosis in some form or another, and, though this is rarer, to the presence of an undiscovered empyema. All those forms of organic disease may be the cause of serious wasting, and I shall consider them separately in a few minutes.

We come now to the last group of cases in which one finds wasting, those, namely, in which there is **no apparent or discoverable cause.** It is to that group, a comparatively small one, that the term 'marasmus,' or, as it is sometimes called, athrepsia, is strictly applicable. I do not think one has any right to talk of marasmus when one is referring simply to a case of insufficient feeding, or where one is dealing with persistent indigestion, or where there is an organic cause, such as those I mentioned in the fifth group. I think one should reserve the term **marasmus** for those cases where there is wasting for which one can find no sufficient explanation. I saw recently in the post-mortem room an autopsy upon a baby which belonged to this group, and the house physician who brought the organs to me said he was puzzled at being unable to find any evident cause for the child's death. That is exactly what one expects in those cases. You search the body thoroughly after death, but can find no reason at all for the wasting and fatal issue. Naturally, theories have been advanced to account for such an occurrence. There are some who state that if you examine the alimentary canal histologically in those cases you will generally find it to be the seat of microscopic alterations. They assert that you will find mal-development of the lymph-follicles and Peyer's patches, and that you will find also atrophy and catarrh of the glands of Lieberkühn. On the other hand, it is denied strenuously by others that these apparent microscopic findings have any significance at all. They say you will find them as commonly in children who are not wasted, simply as a result of post-mortem change, and if you inflate the bowel of children who have died from other diseases you will find a similar alteration brought about purely mechanically. There is perhaps more to be said for those who maintain that this condition of apparently causeless wasting is not due to anything on this side of the alimentary canal at all, but that it is a tissue vice, a failure of assimilative power on the part of the tissues generally, perhaps on the part of the liver particularly. I confess that that sounds rather vague, and I admit it is vague, but I do not know of any way in which to put it more clearly. And such a condition is not unthinkable. You can find analogies for it in certain diseases of grown-up persons. There is the failure of assimilative power with regard to carbohydrates in diabetes; you know of a similar failure as regards fat in some cases of obesity, where the fat is stored up instead of being consumed. There is also the failure of assimilative power with regard to some nitrogenous compounds in gout. And it is possible that some children come into the world with congenital inability on the part of their cells to assimilate nutriment properly; and the conditions under which many marasmic children are born would conduce to that congenital feebleness. They are often born at the end of large families, when the reproductive power may be assumed to be on the wane, or to be partially exhausted, and they have often been brought up under very unsuitable hygienic surroundings, and probably have been always badly fed.

So much for the pathology, if one may call it so, of this condition. The symptoms I need hardly describe to you. The chief one is persistent loss of weight, and in the group of cases which I am now particularly considering—those to which the term 'marasmus' is strictly applicable—there need be no other symptom at all; no special disorder of digestion, no diarrhœa, and no vomiting. The child simply goes on losing weight, with varying rapidity; he becomes shrunken and emaciated, and finally presents that picture of extreme feebleness and atrophy which is so commonly seen in the out-patient room (Fig. 6).

The **treatment of marasmus** you will find by no means satisfactory. The treatment of wasting due to many of those other causes I have mentioned is quite easy. It is easy to increase the amount of food, or to alter it. It is often easy to correct indigestion, or even to deal with some forms of organic disease, but it is not easy to improve the assimilative power in one of these marasmic children. You will find that you exhaust all your ingenuity in devising mixtures of artificial foods without any success; and, indeed, these are the cases which are brought to you with the history that they have had all sorts of foods without any benefit. If you can, you will probably find that the best thing to do with those marasmic children, assuming that they are not being fed from the breast of the mother, is to get them a wet nurse. They certainly do better on human milk than on anything else, and you will save a certain proportion of them by having recourse to that means. Failing that, you often have to use the most easily digested mixtures which you can find. For instance, peptonized milk may be used, and by extreme care in feeding, and by husbanding the child's strength at the same time—that is to say, by keeping him very warm, so that his expenditure of heat is comparatively small—you may sometimes be able to tide him on until the period when the first teeth are cut. Fortunately, about that period the digestive power of such children often appears to undergo a sudden alteration for the better, and towards the sixth month they begin to be able to deal, not perhaps with milk, which in these cases often presents a great difficulty, but they begin to be able to digest starchy foods, and thrive upon them; and after that all the difficulties which you have been combating may disappear. But you have to be prepared for frequent disappointment, inasmuch as these children often go persistently downhill and die, no matter what you do for them. One peculiar symptom which you are likely to meet with towards the termination of these cases of marasmus is that the temperature, which has been previously normal, or perhaps subnormal, rushes up to a very high point. I have known it go up to 108° F., and even higher. This development of hyperpyrexia in the last hours of life is a thing which occurs not uncommonly, and you may perhaps think, on that account, that there will be signs of tuberculosis after death, but you do not necessarily find such signs at all. I do not know what it means, but it is always a symptom of grave significance. I have never known a case which developed it survive more than a few hours at the most.

CONGENITAL SYPHILIS.

I will now pass from the subject of marasmus to consider these two organic diseases, congenital syphilis and tuberculosis, which so often manifest themselves chiefly by the symptom of wasting. We shall take **congenital syphilis** first. I shall not deal at all exhaustively with it, and I shall not describe it as it occurs in older children. I want merely to deal with congenital syphilis as it occurs in young infants, and particularly to tell you how you are to recognize it, because upon its recognition, I need hardly say, the proper treatment of that group of cases in which the wasting is induced by the existence of the syphilitic taint must always depend.

In the first place, if you suspect that congenital syphilis may be the cause of wasting in a case with which you have to deal, you must inquire carefully into the **family history.** I have previously told you that you must inquire carefully into the family history of all infants, but you must particularly do so in those whom you suspect to be the subjects of this disease. You will ask about the health of the previous children ; about the number of still-births, if any ; whether there have been any miscarriages ; and whether the child before you was carried to term. You may get a positive history which is extremely suspicious. You may have a narration of cases of still-birth or repeated miscarriages, or the present child may have been prema-

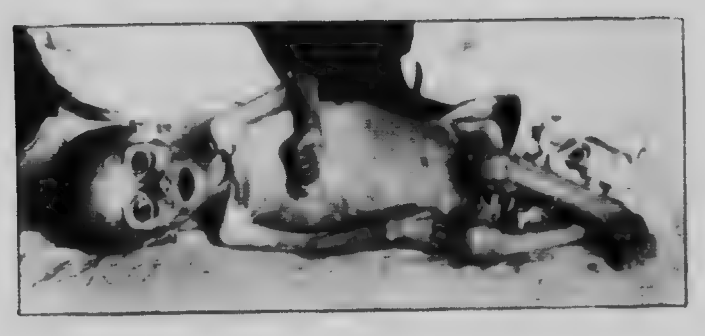

Fig. 6.

MARASMUS.

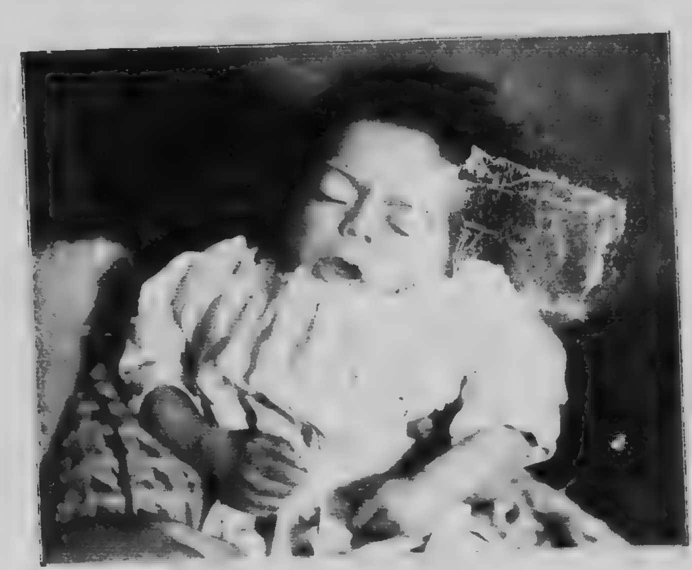

FIG. 7.
THE 'SYPHILITIC WIG.'

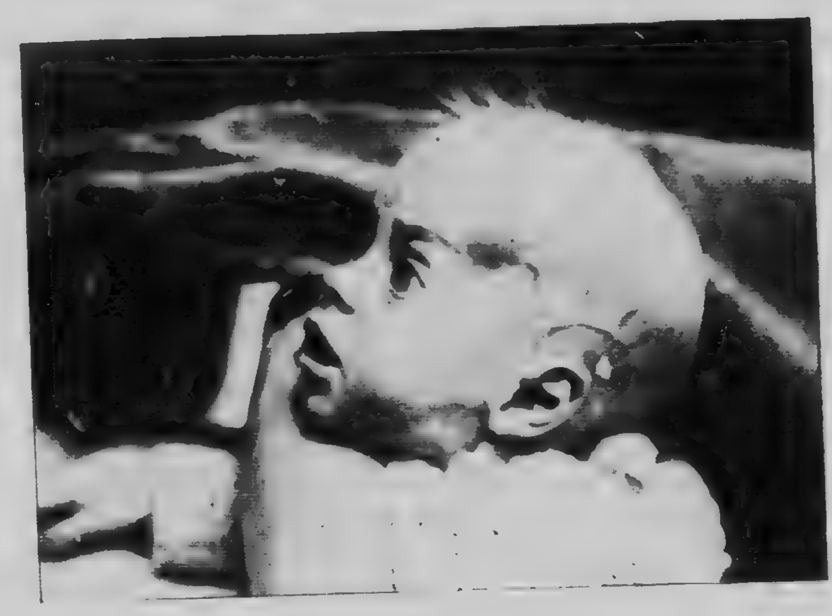

FIG. 8.
ENLARGEMENT OF VEINS OF THE SCALP IN CONGENITAL SYPHILIS.

turely born. But you must be prepared every now and then to hear, in a case in which you may conclude on other grounds that the child is the subject of congenital syphilis, that the family history is good. In other words, you do not always get a suspicious family history, and you must not be led off the scent by its absence. What, then, are the **signs** by which you will recognize the existence of the syphilitic dyscrasia in a young child? You will often, by a mere casual inspection, be able to decide almost at once that the child is the subject of syphilis. There is the characteristic facies which has been often described. The child has a shrunken or wizened up appearance. The skin has a curious earthy tint, which has been compared to that of a mixture of coffee and milk, and the nose often shows a depression of the bridge. There is commonly also an absence or a scantiness of the eyebrows. When you look more carefully at the head you will find frequently, although by no means always a peculiarity of the hair to which I want to direct your special attention. This peculiarity of the hair has never, so far as I know, been described in books on this subject, but you will often find it of very real service in diagnosing congenital syphilis. You will observe in many of these children that the hair is unusually long, that it is straight, fine in texture, and usually dark in colour, suggesting fur rather than hair—a combination of things which one is in the habit of describing as the '**syphilitic wig.**' The existence of this as a sign of congenital syphilis in early life was pointed out to me some years ago by Dr. W. S. Colman, and I have frequently found it of the very greatest value in diagnosing early cases of congenital syphilis. I shall show you immediately lantern slides illustrating examples of the syphilitic wig, in order that you may be able to recognize it afterwards (Fig. 7).

You will find **signs in the bones** in many cases also; a bossing of the fronto-parietal eminences is a common, but not an invariable, sign. The existence of those areas of softening behind the ears and along the occipital suture, to which the term 'cranio-tabes' is applied, is frequently to be made out. You will often find also that there is a slight degree of hydrocephalus, and that the veins on the scalp are prominent and distended (Fig. 8). Along with that you may observe a curious ridging of the edges of the sutures, and a slight thickening round the margin of the anterior fontanelle, the bone being, as it were, slightly heaped up. I do not say that such heaping up is invariably due to congenital syphilis; but, like head-bossing and cranio-tabes, I think it is more commonly due to that than to anything else. Then, passing down over the child, you will frequently find the existence of an **eruption**; but here, again, not necessarily always. Nor is the eruption of congenital syphilitic disease always easy to recognize. It occurs, as you probably know, more commonly about the nates than anywhere else. But you will remember that in that region also eruptions of the nature of intertrigo, due to irritation by the stools or by the urine, are not by any means uncommon, and you will not always find it easy to tell when you are dealing with a syphilitic eruption and when you are dealing with one which is due to this irritation. But there are certain points which will help you. One is the colour of the eruption. In congenital syphilis it has that coppery tint which is so characteristic of syphilitic skin lesions wherever found. You will find also that whereas an intertrigo rash appears only on the opposed surfaces, those which touch one another, a syphilitic eruption tends to wander down over the limbs, perhaps over the calves, and tends to appear on the soles of the feet or the palms of the hands. And when it appears in these situations it often has a silky or glistening look. Condylomata may also be met with, but rarely below the age of one year (Fig. 11).

Snuffling is another common sign, as you know, but I would warn you not to attach too much importance to it. Snuffling and the presence of an eruption are signs which are apt to be overrated as a means of diagnosing syphilis, for those two signs in children are not by any means always due to that disease. Moreover, many syphilitic children do not have snuffling at all. Other causes of it are the presence of a mere cold in the head, or the existence of adenoids. I shall have a good deal to say in a subsequent lecture upon the subject of adenoids

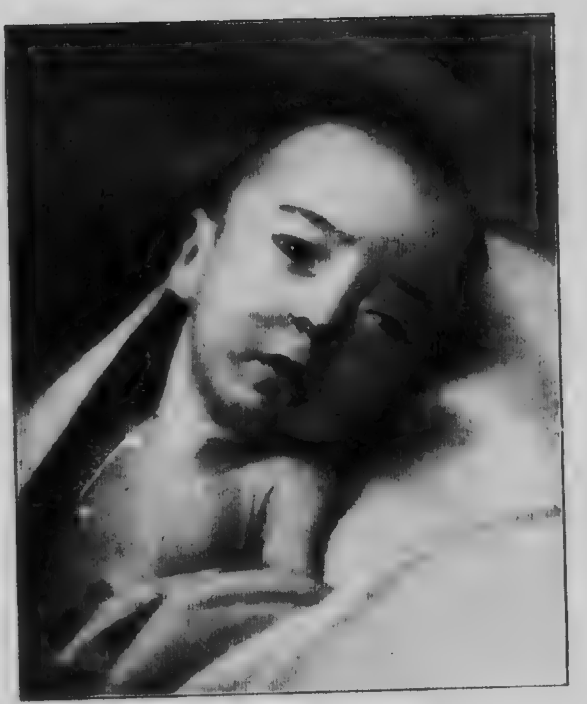

FIG. 9.

CONGENITAL SYPHILIS, SHOWING SCARRING ABOUT MOUTH.

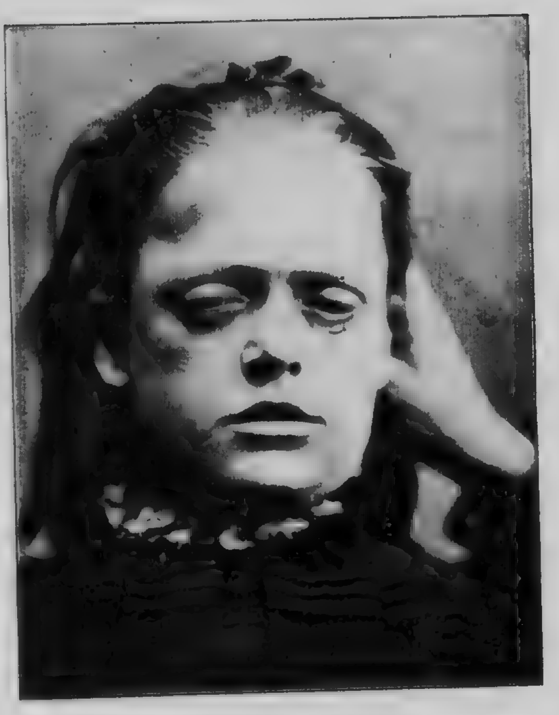

FIG. 10.

FACIES OF CONGENITAL SYPHILIS.

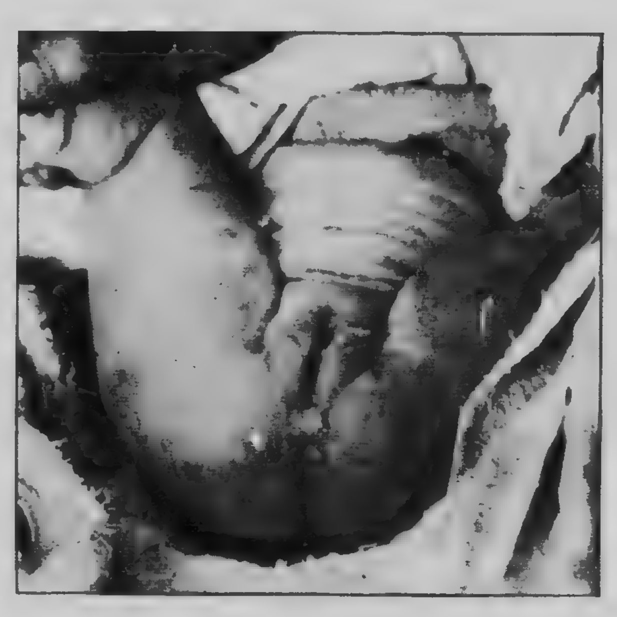

Fig. 11.

CONDYLOMATA.

in children ; and I only want to point out now that adenoids do occur in infancy, and that they may give rise to snuffling and discharge from the nostrils which you may mistake for the signs of syphilis. A cold in the head you should not mistake for it because the cold is usually of shorter duration, whereas the snuffling of congenital syphilis lasts for some time. The snuffling of syphilis does not begin immediately after birth ; there is usually an interval of at least two or three weeks, and it is always accompanied, sooner or later, by the discharge of a yellowish irritating fluid from the nose.

There are certain other symptoms which you may look for. One is the existence of thickenings of the phalanges—what is called **syphilitic dactylitis.** You will find that of help in the diagnosis in a few cases ; and I have also known cases recognized by the existence of gummatous deposits, particularly gummatous epididymitis, which may occur in quite young infants. You will perhaps get some help from an examination of the viscera. You may possibly find **splenic enlargement.** This occurs in about 50 per cent. of cases of congenital syphilis, but you must not expect to find it great in degree. A spleen which has been enlarged by congenital syphilis is not often very big ; it is palpable, but usually not more than that. An affection which also occurs sometimes in this disease is an inflammation of the ends of the long bones, so-called **syphilitic epiphysitis.** It is really a perichondritis affecting the whole length of the bone, but the tenderness and swelling are most marked round the joint, because the muscles are inserted there and drag upon the perichondrium. It usually affects the upper limbs. On account of the tenderness there is a loss of ability to use the limb, sometimes spoken of as syphilitic pseudo-paralysis, so called because it is not a genuine paralysis, but an inability to make use of the limb from the existence of pain. I shall have more to say about that when I talk about paralysis in infancy generally.

Last, I would mention a symptom which has been emphasized by Dr. Eustace Smith, and which one sometimes finds of use in diagnosis, and that is **restlessness at night.** Syphilitic children, for some reason or other, are apt to be wakeful and restless at night, and to cry a great deal at that time. Dr. Smith suggests that this may be due to the existence of periosteal pains, just as there are often pains in the bones of adults who suffer from syphilis. At all events, you will sometimes find that this symptom will put you on the right track, and enable you to diagnose the existence of a syphilitic taint.

When all is said and done, and you have all these possible signs before you, there will still remain a residuum of cases in which you must be in doubt, because there are many children who waste merely because they have a slight syphilitic taint; they are not, strictly speaking, sufferers from congenital syphilis, but there is a syphilitic strain in the family, and there is enough of the poison in them to affect their nutrition adversely; and it is in these cases that one often has got to be guided in the diagnosis by the results of treatment, by giving antisyphilitic remedies, and seeing what happens. You will remember I showed you some charts of wasting infants, in whom the administration of gray powder at once caused a gain in weight, and in a certain proportion of these cases that result is probably due to the existence of a congenital syphilitic taint which one has overlooked. I told you that it is not invariably due to that. I think one sees gray powder cause a gain in weight in cases in which one has every reason to exclude syphilis; but in some of the cases the explanation of such a rise in weight may be that the child has been slightly affected by this disease.

If you have satisfied yourself that wasting is due to congenital syphilis, there is a definite line of **treatment** marked out. You have to treat the syphilis by mercury, and the best way of doing so is by the mouth, giving ½ grain of gray powder in this way four times a day. Begin with ½ grain in the morning and ½ grain at night, until you have got up to that dose four times a day. It is important to recognize signs of overdosage. How shall you know when you are giving too much mercury? You know that you can recognize overdosage in adults by the occurrence of salivation, but infants never salivate with mercury. I do not know why that is, but it is probably due to the fact that the salivary glands do not develop well until the teeth are cut. At all events salivation is not to be looked for when giving mercury to young children. The great sign in infants is the appearance of diarrhœa. So if during your treatment with mercury diarrhœa develops, remember it may be due to the giving of too large doses.

How long shall you continue to give mercury? That is a question which often presents real difficulties in practice. Many people advise you to stop giving mercury when all the symptoms have disappeared. I think those who recommend you to continue the treatment for nearly a year, in at least moderate doses, are probably better advised. There is a danger in stopping too soon. Over and over again it has been my experience to stop mercury when everything seemed to be going on well, and to have the child brought back a month or so later with definite signs of syphilis again. Of course, you often find it difficult to persuade mothers to allow treatment to be carried on. The tendency in hospital practice is for patients to cease attending when the symptoms have disappeared, so you must impress upon parents at the outset the necessity for prolonged treatment. Sometimes you will find it difficult to give mercury by the mouth ; there are some children in whom even quite small doses produce diarrhœa, and in these cases inunction into the skin is a good method of treatment. I generally use blue ointment, about ½ drachm of it being rubbed into the abdomen once, or at most twice, in the day, after which the skin is covered by a flannel binder, so that gradual absorption of the ointment goes on. You will find syphilitic children tend to suffer from a sort of cachexia and from anæmia, and if that is a prominent symptom it is advisable to give cod-liver oil and iron as well as mercury. Indeed, there is reason to suppose that the prolonged administration of mercury is in itself apt to produce anæmia, and that is probably why many people advise its early discontinuance. So when you go on giving mercury after the symptoms have disappeared, you may find it well to give iron and cod-liver oil in addition.

What about the **treatment of local lesions?** I think you should treat the snuffles by syringing or brushing out the nose with a solution of borax or of corrosive sublimate (1 in 2,000), and putting a weak mercurial ointment up the nostrils by means of a feather. That prevents the crusts from drying and the nose from becoming blocked up. I usually employ weak white precipitate ointment for the purpose. The other chief local symptom which one has to treat is the eruption, and that you can do either by weak corrosive sublimate baths or by dusting powders containing calomel. Sometimes you may use a combination of weak white precipitate ointment and zinc ointment.

LECTURE X

TUBERCULOSIS IN CHILDHOOD

Gentlemen,—In the last lecture we were considering wasting as a sign of disease in infancy, and we saw that there were several morbid conditions which may be associated with it as one of the chief symptoms. I enumerated to you several possible causes of wasting in children which you must consider when a case comes before you, and I dwelt particularly upon congenital syphilis as one of the organic diseases which may be a cause—sometimes an unsuspected one—of that condition. Now, in this lecture I wish to direct your attention to the other great organic disease which may be a cause of wasting in children, both in infancy and in later childhood. I mean tuberculosis.

The **frequency** of this disease in children will be realized from this fact : that careful hospital statistics, taken in various parts of the world, show that about one-third of all the children who die in hospital die of tuberculosis in some form or another ; and, further, in about 12 per cent. of the remaining cases tubercle is present in a latent form. If you examine the bodies of those children after death, you will find that the tuberculosis tends to attack one particular set of tissues with a very striking degree of regularity. Those are the tissues which are concerned with the formation of the blood. Ninety per cent. of all the cases show an affection of the lymphatic system or the bones. The lymphatic system, you will remember, is engaged in the formation of one variety of white blood corpuscles, and the bones are quite as much blood-forming organs as they are supporting tissues. And so when one says that the bones are affected, one really means that a blood-forming organ is affected. And I want to impress upon you this fact, that it is this particular set of tissues which, more than any others, tends to be involved in tuberculosis in early life. If you look at the diagram on the wall, which shows the age distribution of 500 consecutive cases of tuberculosis in children, taken from the statistics of the Great Ormond Street Children's Hospital by Dr. Still, you will see that tubercle attains its maximum frequency in the second year of life. And I want you to correlate in your minds those two facts—first, that it is the blood-forming tissues that are specially affected by tubercle; and, secondly, that it is in the second year of life that tubercle attains its maximum frequency—because I think there is a relationship between them which is not an accidental one. I think it is probably owing to the blood-forming organs being in a very active state of vitality at this period that explains their liability to tuberculosis. When I come to speak of the anæmias of infant life I shall have occasion to point out that the blood of children undergoes a very important transformation in character at just about this time, towards the end of the second year. It is about that time that the tissues which form the white blood corpuscles come to attain their greatest degree of activity. After that the lymph-forming organs gradually pass into the condition in which they are found in the adult.

Of more importance for us, however, is it to study how the tubercle bacillus most commonly finds access to the body in childhood—*i.e.*, its **path of infection.** It is all very well to know that the tissues most involved are the lymphatic or blood-forming organs; but what we specially want to know is how the bacillus gets there at all. Of a series of 216 cases which were carefully studied from this point of view by Dr. Still, it was found that the access of the bacillus had been through the lungs in 63·8 per cent., through the intestine in 29·1 per cent., and through the middle ear in about fifteen cases. These statistics were derived from the Great Ormond Street Children's Hospital, but there are similar statistics from other children's hospitals. They all tend to show the same thing—namely, that the lung is by far the commonest path of infection.

You will see at once what the significance of that is. It means that the talk about the milk of tuberculous cows being the chief cause of the infection of children with tubercle is certainly wrong, and it also means that dust must be regarded as the main source of infection in the child just as it is in grown-up people.* The tubercle bacilli are conveyed into the lungs, probably mixed up with dust, and thence they find their way into the lymphatics. When one speaks of the lung in this connection it must be remembered that the chief part affected is the bronchial glands at the root of the lung. But these are not likely to be affected if they are perfectly healthy. Unless there be a great inherited predisposition to tubercle the healthy gland may be assumed to be able to deal with such tubercle bacilli as find access to it; otherwise all of us would be suffering from tuberculosis. And the reason why the bronchial glands in children become so frequently affected no doubt is that they are very often not in a healthy condition; they have already been the seat of inflammation. How do they become inflamed? They become inflamed secondarily to catarrh of the bronchi or alveoli. In other words, the bronchial glands become inflamed during an attack of bronchitis or broncho-pneumonia. The bronchitis or broncho-pneumonia is recovered from, but the glands are left in an unhealthy condition, dust containing tuberculous material is inhaled, and the bacilli start to grow in the glands and set up a caseous degeneration of them, which is the starting-point of tuberculosis. So I want to impress upon you, as the great predisposing cause of tubercle in children, that group of diseases which are accompanied by catarrhal processes in the bronchi and lungs, particularly such diseases as measles and whooping-cough. And you will constantly find in your practice that children will be brought to you for wasting and some vague complaint of ill-health, and on inquiry into the past health, you find that they have had an attack of measles some months or weeks before, and have not been well since. Then when you come to make a physical examination, you will find that they are the subjects of tuberculosis. That is a very common history.

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* This statement will probably require modification, for evidence is accumulating that bacilli which have gained access to the body by the alimentary canal may travel up to the bronchial glands and first reveal their presence there.

Now, no matter by which path the bacillus finds access, no matter whether it is through the bronchial glands or through the intestines, affecting the mesenteric glands or through the middle ear, it sooner or later **generalizes** itself. And that is the other point I want specially to emphasize, —that no matter what the primary path may be by which the child becomes infected, the disease has a great tendency to become widespread, a much greater tendency than is the case in grown-up persons. What usually happens is that one of these caseous bronchial glands softens; it ulcerates its way into a bronchus, and the caseous material gets aspirated into the lungs, and so pulmonary tuberculosis is set up. In other cases perhaps the infection gets conveyed by ulceration into a blood-vessel and is disseminated in that way through the body. At all events, in whatever way dissemination occurs, it takes place very quickly. That is why it is so important for you as practitioners to diagnose tuberculosis in children as early as possible, because, unless you recognize it early and treat it promptly, it will certainly before long become generalized, and then it may be too late to treat it at all.

That leads us naturally to consider how you are to **diagnose the existence of tuberculosis** in children. I shall consider first of all the diagnosis of *general tuberculosis*. We will assume that the process has already become generalized. How are you to know, then, that the wasting in this particular case is due to general tuberculosis or not? Often, I believe, it is not possible for you to know at all. It is not possible to know until the post-mortem in many cases. The diagnosis of general tuberculosis in children is extremely difficult, and it is so for this reason: that the process kills the child before the tubercles have had a chance of breaking down. You know that unbroken-down tubercles do not give rise to physical signs. You may have a lung stuffed with miliary tubercles, but until they soften and break down you have no means of telling that they are there, and it is on this account chiefly that the diagnosis is often so uncertain. There are, however, certain symptoms and signs which should make you suspicious. One of these suspicious symptoms is the occurrence of irregular pyrexia along with wasting for which you can find no other cause. Again, if there is much apparent illness and fretfulness on the part of the child, without obvious or apparent reason you should be suspicious that it is a case of tubercle. But there is no other symptom to help you much. You will easily see that the danger you run is of attributing those symptoms to, perhaps, a slight dyspepsia, or catarrh of the stomach or intestine ; or, perhaps, to a slight bronchial catarrh, or to some trivial condition of that sort which may be present along with the tuberculosis, and not to tuberculosis itself. It is so extremely difficult to assign its proper value to each of these symptoms that you have often to suspend your judgment.

Amongst the **signs** which may give you some assistance in the diagnosis of tuberculosis are certain appearances in the child—appearances which clinical experience has shown to be often associated with tubercle. Tuberculosis is known to be commoner in children of a certain type. You know the fragile, fairy-like type which is supposed to be particularly marked out for tuberculosis. That is an extreme example of what I mean. In young babies, however, that type is not so noticeable, and you have to go by such minor indications as long eyelashes, or the presence of an excess of downy hair, especially on the back between the scapulæ. There is also, which you will find of value, a tendency to the appearance of blotches of erythema upon the skin on very slight pressure or irritation. These signs may perhaps seem to you trivial, but very often, taken together and along with the family history and the general symptoms, they may suffice to put you on the right track. Of course, you will look also for signs indicating the starting-point of the process. First of all you will look for signs of the disease having started in the lungs. I shall describe to you immediately what the signs associated with tubercle of the bronchial glands are ; but I shall only say here that in this case again you have great difficulty in coming to a right judgment, because, admitted that you get moist sounds over the lungs in a case which you suspect may be one of tuberculosis, you are still in doubt as to whether that is due to breaking-down of tubercles or not, because there is nothing commoner in marasmic children than for them to suffer from a slight attack of hypostatic pneumonia as they lie in bed, and that may easily give you moist sounds over the back. So it is only when you get signs of breaking-down on the anterior aspects of the lungs that you should attach much importance to them.

I next pass to the consideration of the physical signs associated with **tuberculosis of the bronchial glands**; in other words, the signs which you find when the tubercle finds access, as it usually does, by means of the lungs, and when it has not yet become generalized. Here, again, there is no quite conclusive symptom. There will be more or less wasting, and perhaps there will be irregular pyrexia. There will also be general fretfulness and illness. But the only local symptom of any value is the occurrence of a spasmodic cough. Children with enlarged bronchial glands often suffer from such a cough, which you will be apt to mistake for whooping-cough, which it may simulate very closely. Many cases of whooping-cough which are supposed to drag on for months are probably cases which may have begun in whooping-cough, but which are now suffering from secondary enlargement of bronchial glands. Note, then, that one point which will help you is the existence of this spasmodic cough. When you come to the physical signs you will find that they are only present in a minority of the cases, and you have no difficulty in seeing why that should be. The bronchial glands are not very large; they lie deep down in the mediastinum, and it requires a great deal of enlargement of them to produce signs which you can recognize with certainty. So you must be prepared to find that in cases in which after death you are able to recognize a considerable degree of enlargement of the bronchial glands there were no signs of them during life. Still, in some cases, when the enlargement is very great, it will reach the surface, and the chief sign in such a case is dulness behind the manubrium sterni—dulness which extends outwards towards the apices of the lungs and downwards towards the heart. I would warn you, however, of one possible fallacy here—namely, that in enlargement of the thymus children may closely simulate this sign. There is no way of discriminating exactly between the two. Another sign which you will find is distension of the veins in the neck. This is due to the fact that the enlarged bronchial glands tend to press upon the superior vena cava. Further, owing also to such pressure upon the veins, there may be a bruit heard on listening over the manubrium. Dr. Eustace Smith has pointed out that this is best heard if the child stretches its head back, which causes the superior vena cava to be pressed upon by the mass of glands. But I do not think you should attach too much importance to that sign. One certainly hears a bruit of that sort in many cases in which there is no reason to suppose that the bronchial glands are enlarged. On the other hand, its absence is an indication of some value, which should make you suspicious that you are not dealing with such enlargement. I am afraid these are the only signs which one can give you as indicative of enlargement of the bronchial glands, and you will see that, taken altogether, they are not of very great value. What I wish to impress upon you by all this is the great difficulty which you will have in making up your mind about either bronchial tubercle or general tuberculosis; and you must often remain in doubt, and must not blame yourselves too readily if, after death, you find extensive tuberculosis when you have been regarding the case as one of ordinary wasting.*

The next form of the disease which we come to is much more hopeful, both as to diagnosis and treatment—namely, **tuberculosis in the abdomen**—those cases in which the bacillus has gained access through the alimentary canal into the intestinal glands and peritoneum. Abdominal tubercle differs so much from tubercle elsewhere in children —and the same is true of grown-up people—that one is tempted to suppose it is a different disease ; and it has recently been suggested that it may prove to be bovine tuberculosis in man, and therefore a milder form of a similar pathological process. If that were established, it would at least explain the very marked clinical differences between abdominal and other forms of tuberculosis. When we come to consider prognosis and treatment, you will find that abdominal tuberculosis is much more curable than any other form ; indeed, one takes quite a cheerful view of it, because a large proportion of cases get perfectly well.

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* The cutaneous tuberculin reaction is now often employed as an aid in the diagnosis of tuberculosis in childhood. It is, however, by no means a certain test. Latent or healed tubercular lesions are so common in childhood that a positive reaction is only of value in quite young children. A negative reaction is more trustworthy as excluding tubercle, but it must be remembered that a negative reaction may be obtained if the lesion is very extensive or active.

Abdominal tuberculosis resembles the other forms in being most common in the second year of life. The statistics of a number of cases of fatal tuberculous peritonitis in children were collected by Dr. Still when he was Registrar of the Great Ormond Street Children's Hospital. He found that the maximum frequency of death occurs in the second year. That is against the milk theory. Seeing that children live almost exclusively on milk up to the first year of life, one would expect abdominal tuberculosis to be common in the first year if milk were the main source of infection. Not only does abdominal tuberculosis occur most commonly in the second year of life, but at that particular time the abdominal disease is usually only part of a general tuberculosis. In young infants it is not purely abdominal tubercle which you have to deal with. You may think when you examine the case that it is abdominal tubercle only, but you will almost certainly find that there are tubercular deposits elsewhere. That is only another way of saying that tuberculosis in young infants, no matter where it starts, tends rapidly to become generalized, and if it starts in the abdomen it ends by involving the lungs or the meninges of the brain ; and what you will find constantly happen in these cases is that the case drags on until of a sudden it flares up into tuberculous meningitis, which is followed by death.

There are two **varieties of abdominal tuberculosis.** There is the plastic form and the ascitic form. Most of you must be quite familiar with those two varieties. The plastic form consists in an enlargement of the glands and a matting together of the coils of intestine with tubercular deposits in the peritoneum and mesentery. The ascitic form is that in which an effusion of serous fluid takes place into the peritoneal sac.

To the first or plastic form the term **tabes mesenterica** is sometimes applied, but I think quite erroneously. I strongly advise you to discontinue the use of that term altogether. For my own part, I have hardly ever seen a case of true tabes mesenterica. It is a disease which, if it occurs at all, must do so with extreme rarity. Rightly used, the term 'tabes mesenterica' means an enlargement of abdominal glands, with caseation, and nothing else. That may occur, and probably does occur sometimes, but it is very unusual. It will certainly conduce to clearness if we talk merely of the plastic type of abdominal tuberculosis. This type is commoner than the ascitic. Some people say that it is ten times commoner, but my own observation would not corroborate that, I think. In older children at least the ascitic form is certainly commoner than that; it is much more than one-tenth of the other. You will recognize this form by the general signs of tubercle, the wasting, pyrexia, and so on. The local signs are hardness, with nodulation, in the abdomen.* You may take it as a broad general rule that if you find nodules in the abdomen of a child, if you can exclude fæcal masses, the probabilities are enormously in favour of those nodules being tuberculous, no matter where in the abdomen they are situated, and no matter what they feel like. Tubercle is so very common compared with anything else that you do wisely to regard every mass in a child's abdomen, where you can exclude a fæcal accumulation, as being a tuberculous deposit of some sort. You can easily exclude or confirm a fæcal accumulation by the administration of purgatives and enemata, and if you are dealing with a doubtful case of abdominal tuberculosis you should take that precaution, seeing especially that the large bowel is thoroughly emptied. Then examine the abdomen again, if need be under an anæsthetic, and see if you can still discover the lumps. They are due partly to caseous deposits in the mesentery and elsewhere, partly to glandular enlargement, and partly to coils of bowel bound together by adhesions, and are the main physical sign of the plastic form of abdominal tuberculosis.

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* In many cases also the thickened and rolled-up omentum can be felt as a band crossing the abdomen transversely above the level of the umbilicus.

The **ascitic form** you recognize by the presence of fluid. Is the ascitic form of tuberculosis ever simulated by any other peritoneal effusion? In other words, may there be clear fluid in the abdomen of an infant which is not due to tubercle? Of course, such effusions may sometimes occur in the course of cirrhosis of the liver, and you may find a difficulty in excluding the presence of that disease. Not long ago I had the case of a little girl in whom that problem came before me in a very acute form. She looked the picture of robust health, but her abdomen was full of fluid, and the liver was about two fingers' breadths down. Its edge was also very hard. It was thought to be a case of cirrhosis, and that it would be well to treat the case from that point of view. However, we watched the patient, and she developed a temperature, and then we began to be suspicious, and thought it would be better to operate, because if it were tubercle it would be the right thing to open the abdomen; and even if it were ascites due to cirrhosis, operation would not do any harm. On the day decided upon for operation she suddenly became comatose, developed all the signs of tuberculous meningitis, and died; and, post-mortem, the abdomen was found to be the seat of very generalized tuberculosis. Such a case may cause you difficulty; but I would again remind you of the great frequency of tuberculosis and the comparative rarity of cirrhosis, so it will be safer when in doubt to diagnose tubercle. Clear effusions into the abdomen due to simple chronic peritonitis have also been described, but they are very rare. They are said to occur after measles or scarlet fever, or after injury. Many good observers, however, are of opinion that all clear effusions of an inflammatory sort into the abdomen in children should be regarded as tuberculous.

Both forms of abdominal tubercle are commonly accompanied by **ulceration of the intestine,** according to some statistics in as many as 70 per cent. of all the cases. Ulceration does not necessarily show itself by clinical symptoms. It does not necessarily cause diarrhœa. But where ulceration becomes extreme and extensive you will find it tends to be accompanied by an extreme degree of anæmia—a degree which may almost resemble pernicious anæmia in its intensity.

We turn now to the **general treatment of tuberculosis**; but one may first say a word about prevention, which, of course, is more important than cure. I need not remind you of the general hygienic rules which should be observed in the rearing of children: attention to proper feeding, proper exercise, proper ventilation of the sleeping rooms, and all those other commonplaces which even lay people now recognize. But I would direct your special attention to the question of dust, because if dust is the chief cause of infection, as statistics show it to be, then dust must be one of the chief things to fight against. Another point is this: seeing that dust infects through the bronchial glands, what you have to do if you want to prevent children becoming tuberculous is to keep their bronchial glands healthy. To do that you have to treat every case of catarrh of the lungs or in the bronchi seriously. You must not think that even a slight bronchitis is a trivial thing, nor yet a little attack of broncho-pneumonia. So far as you can you must see that convalescence is made thorough and complete; that there is not left behind a little wheezing, or a few crepitations at one base. You must try to restore the child to complete health. As it is certainly possible for the abdominal form of tuberculosis to be conveyed by the milk of tuberculous cows, you should, of course—particularly in the case of children who are predisposed by heredity or by surroundings to become infected with tubercle—see that the milk which they get is at all events boiled. I do not say it should necessarily be sterilized, but it should be boiled.

With regard to the **treatment of different forms** of tuberculosis, I need hardly say there is no special treatment for the generalized type. Whether general tuberculosis is ever recovered from at all it would be very hard to say. Such a high authority in the matter as Sir William Jenner was of opinion that general tuberculosis is recovered from in children much more commonly than is supposed. It depends a good deal upon what you mean by generalized. No doubt deposits of tubercle in the lungs and abdominal organs may apparently be recovered from ; and years afterwards you may find evidence of it in calcification of the glands. But once tubercle has become really generalized, I think anything which you can do in the way of treatment is not likely to influence the process much. The same is true of tubercle of the bronchial glands, which, merely from its situation, it is difficult to treat, for you cannot get at the bronchial glands. Not even the modern surgeon has yet attempted to excise them. For practical purposes, therefore, your treatment in both general and bronchial tuberculosis must resolve itself into simply putting the child into the best possible hygienic conditions, in the hope that the natural powers of resistance may be able to bring the disease to a favourable termination. In a case of **abdominal tuberculosis,** on the other hand, you can do a great deal by treatment. There are no cases of tuberculosis which you approach with more hopefulness than those of abdominal tubercle in children ; and I should say that the prognosis will be better in older children than in younger, for the reason that in young infants tubercle generalizes so fast that you do not often have to deal with the local disease only. In older children generalization occurs more slowly, so that you have a chance of getting it in time, and treating it successfully. The first thing to do is to attend to the general surroundings of the child. Such children should be sent away, if possible, out of towns, and be taken to a place like Margate, and spend their time by the seashore in a wheeled carriage, lying down. The abdomen should be supported by a broad and firm binder. You will find it an advantage to apply iodoform ointment locally ; it should be rubbed well into the skin of the abdomen. I also often use, and I think with advantage, liniment of mercury, ½ ounce or so being rubbed in night and morning all over the abdomen. Above all, make it your first care to see that the stomach and bowels are in good order, so that the child can take and digest a suitable quantity of food. You must correct gastric disorder if it exists, and do all you can to increase the appetite. If there be diarrhœa, you must make a point of checking it before you do anything else. Having done that, you can proceed to feed the child up, seeing in particular that he has plenty of fatty foods. With regard to medicines, you will find help from cod-liver oil, iodide of iron, and creosote. You see there is nothing in this line of treatment other than what your common-sense would dictate. But you have often to consider in those cases the question whether you are to continue medical treatment, or whether you are to have recourse to surgical aid. I do not think it is often advisable to have recourse to surgical treatment in those cases right away. I think the necessity for **surgical treatment** in abdominal tuberculosis in children is very much exaggerated. Taking the last six cases of purely abdominal tuberculosis which have been under my care simply as they turned up in the out-patient room, and all between three and nine years of age, I had the abdomen opened in only one of them, and that child recovered, although it suffered for a time from fistula of the umbilicus. But the other five children all recovered completely without operation at all. So you see medical treatment is of very considerable use. By recovery I mean that, at the end of two years, some of them now at the end of three years, they are apparently in perfect health. You should speak cautiously of recovery from abdominal tuberculosis. I do not think one has a right to say that a case is completely cured simply because after a few months the symptoms have not recurred. The symptoms in this disease are apt to remain latent for a considerable time. Such a child may, perhaps, get an attack of diarrhœa, or other slight illness, which pulls him down, and, his resistance for the time being lowered, the process flares up again. Seeing, then, that medical treatment gives such good results, what should guide you in having recourse to operation ? In the first place, all cases of abdominal tuberculosis should get the benefit of operation if fever and continuous wasting persist in spite of the medical treatment I have sketched out for you. The continuance of such symptoms means that medical treatment is not going to accomplish much, and it is well to give the patient the benefit of operation although it must be confessed that many such cases, perhaps the majority, do not get well under surgical treatment either. Further, you will always be biassed in the direction of operation if you are dealing with the ascitic form as opposed to the plastic, because the ascitic cases certainly do better under operation than those in which the disease is mainly in the glands or the peritoneum. Numerous statistics bear out this statement. For instance, the records of the general hospital in Heidelberg, embracing cases at all ages and not only in children show that of forty-one persons operated upon for the ascitic form of tubercle, twenty were completely cured—that is to say, about 50 per cent. Of nineteen cases of the other form, the plastic, only about four could be described as cured. So you see the ascitic form certainly offers a better chance than the other of successful treatment by means of operation. On the other hand, you must remember that the total statistical results of surgical operation are not so much better than the medical statistics if you take them all round, and so it is as well to give every case the benefit of thorough medical treatment first, especially if there be any objection to operation on the part of the parents. Operation in the ascitic form, properly performed, is almost perfectly safe. In operation on the plastic cases there is a greater risk. Every now and again in such cases, owing to the presence of adhesions, the intestine has been opened into by the operator, and sometimes the bladder also. I have seen the intestine and the bladder both opened into at the same operation. So you see the procedure is not devoid of danger. Still, if medical treatment fails in such a case, I think it is right that you should have recourse to surgery.

LECTURE XI

RICKETS

GENTLEMEN,—I shall ask you to-day to study with me a nutritional disorder, which has the peculiarity that it is found in children only—I mean rickets. On the Continent rickets goes by the name of the 'English disease.' That is not because it is commoner in this country than abroad; indeed, I suppose there is no place where rickets is commoner than in Berlin. But it is called the 'English disease' because the first accurate description of rickets as we know it now was given by an English physician, Dr. Glisson, whose name will be familiar to you in association with Glisson's capsule in the liver. In the year 1681 Glisson wrote a treatise on rickets, which was then apparently a new disease. He described it as having first appeared in the south-western divisions of England, and having spread thence over the country. He thought it began in the south-western districts because these were mostly inhabited by the more luxurious part of the community. Whether rickets has increased much since that time it is a little difficult to say; but I think one can be pretty sure that it has increased, because all those factors which tend to predispose to rickets are such as are daily becoming more pronounced. One of those factors is that inability on the part of women to nurse their own children which I have already deplored in a previous lecture. Another is the increase in women's work. With the advent of the industrial epoch in the middle of last century, and the establishment of factories, there opened up at once a great field for the labour of women. This tempted them away from their domestic duties and from the personal care of their children. That state of things persists, and in many of the large industrial towns rickets is extremely prevalent, for the reason that women go to work in the factories, and their children are left at home badly looked after and improperly fed. A third factor in the production of rickets is found in the tendency for the population to be crowded together in large cities, which is such a striking feature of social life at the present day. Now, so potent are these factors, and so prevalent is rickets in consequence of them, that in many towns more than half the children seen in the out-patient room exhibit the disease in a more or less marked degree. So that the dictum pronounced by Sir William Jenner a good many years ago, when he first began to work at Great Ormond Street, still holds good—namely, that 'rickets is the most common, and, in its indirect results, the most fatal of all the diseases which peculiarly affect children.'

The word 'rickets' is derived from the Norman-French term 'riquets,' which meant deformities, particularly deformities of the spine, or persons who were suffering from such deformities. And it was owing to the fact that one of the main symptoms of rickets is the existence of deformities of bone that this name was first applied to the disease. Glisson, or one of those who wrote with him, proposed as a substitute a sort of pseudo-Greek term, 'rachitis.' That, however, presents no advantages over the name commonly used, which has at least the merit that it conveys to you one of the main symptoms that cases of rickets present—namely, deformities of the bones. It would be quite wrong, however, to suppose that that is the whole of the trouble. You would have a totally erroneous conception of rickets as a disease if you were to imagine that it consists solely in the production of osseous deformities. You must remember that there is a visceral side to the affection as well, which, so far as effects upon life are concerned, is more important than the mere deformities due to alterations in the bones. As the bone deformities, however, are the most prominent and striking change, we shall consider them first.

The **changes in the bones in rickets** are of two sorts.

There is, in the first place, an alteration at the point of ossification, the nature of which has been summed up by saying that there is an exaggerated preparation for ossification and a diminished accomplishment of it. That is to say, the preliminary stages of multiplication of cartilage cells go on in an exaggerated degree, and the cells cease to be disposed in those regular rows which are characteristic of normal ossifying cartilage, and become arranged irregularly. The result of this is that the line of ossification becomes thickened, and in consequence you can observe, clinically, enlargement of the epiphyses. I show you the cast of the wrist of a child who suffered from rickets, in which you can see very well the thickening and enlargement of the epiphyses due to this exaggeration of the preliminary stage of ossification. Such thickening is usually first seen in the ribs. It begins there, or can be earlier recognized there than anywhere else. In consequence there is produced a row of knobs down the sides of the chest, to which the term 'rickety rosary' is applied, because the appearance is like a row of beads. Thickening takes place not only on the outside of the rib, but even more markedly on its inner aspect; and it may so press upon the lung that there is a groove of compressed and solidified pulmonary tissue corresponding to the enlarged epiphyses. The specimen I have here shows extremely well such an enlargement at the ends of the ribs just where the cartilage and the osseous part join. Not only are the bones which are developed from cartilage affected in this way, but a similar process takes place in those bones which develop in membrane. Hence the skull bones undergo considerable thickening at the point where ossification is most active. Here I show you the skull of a child which was the subject of rickets, and in it you will be able to feel that in the centre of the frontal and parietal bones there is a considerable degree of thickening. That is one change which the bones show—enlargement at the point where ossification is going on. But they show in addition another general change—namely, a process of softening. The softening is due to the absorption of the mineral constituents of the bone. If you analyze the bones from a case of rickets and compare them with normal bones, you will find that whereas in health there is [illegible] per cent. of organic and 65 per cent. of inorganic matter, bone in rickets shows quite the reverse—namely, 79 per cent. of organic matter, and 21 per cent. of inorganic. In other words, there has been an absorption or removal from the bone of its mineral constituents—chiefly phosphate of lime. I shall point out to you the significance of this more fully later on, when we come to the theories which have been advanced to explain rickets.

When phosphate of lime is removed from the bone, you can easily see what the consequence must be. The bone bends, and the direction in which it shall bend is determined solely by the line of chief pressure. Now practically the lines of pressure depend upon the position which the child habitually assumes. In the legs, for example, one usually finds that in the femur the curve is forwards. In the tibia there is a sharp bend in the lower third, such as you see in the specimen before me; or another common deformity is for the tibia to be curved outwards, producing bow-leg. These deformities are largely due to the way in which the child sits with his feet tucked under him. It is that which causes the femur to bend forwards and outwards, and causes the tibia to kink forwards in its lower third. In the same way the humerus tends to bend outwards, and so also do the radius and ulna, for a rickety child tends to sit up and lean forward, supporting his weight upon the arms. In this way it is possible to give a mechanical explanation of the direction of bending, and the variations [illegible] them depend simply upon the variations in the [illegible] attitude of the child.

[illegible] groups of changes—the visceral—are of even [illegible] importance than the osseous. I might remind you in this connection of another saying of Jenner, that it would be as reasonable to regard rickets as a disease of bones only as it would be to regard typhoid fever as merely a disease of Peyer's patches. The visceral changes consist first and chiefly of catarrhs affecting the lungs, stomach, and intestines. Secondly, they consist in a tendency to fibroid change in some of the internal organs, particularly

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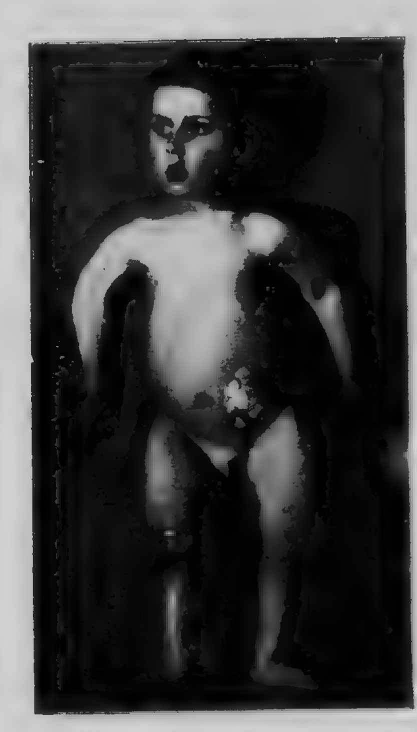

Fig. 12.

RICKETS OF 'OSSEOUS TYPE,' SHOWING CURVATURE OF BONES.

the spleen, with enlargement of that organ, and an increase in the fibrous stroma. The frequency with which enlargement of the spleen is met with in rickets has been considerably disputed. Probably, however, it is not appreciably enlarged in more than 5 per cent. of the cases—that is to say, not enlarged to a degree sufficient to enable it to be felt. These visceral changes have this great importance, that they cause, or tend to cause, death much more than the osseous changes. It is largely a matter of indifference to a child whether the bones are hard or soft, unless he happens to be walking about. But it is a matter of serious importance if he is constantly exposed to attacks of bronchitis or a catarrh in the intestines, because these repeated attacks of catarrh subject the child to a great risk of the development of tuberculosis.

Now, to pass to the more clinical side of rickets, it is well that you should recognize certain distinct **types of the disease.** First of all, there is the acute type, which some people call '**acute rickets.**' But this term is one which it is better to avoid, because many cases of infantile scurvy have been described under it. But in spite of that there *is* a group of cases which one can class together as showing a rapid onset of the disease and a great tendency to the development of visceral rather than osseous symptoms, and as being characterized by considerable tenderness of the bones, as well as a great tendency to sweating. Such children sweat profusely about the head, particularly when they are asleep, and also about the trunk—so much so that you will often be able to recognize them in a children's ward by the way in which they kick off the bedclothes. In such cases I think you will find that the disease has come on rather rapidly. There is a second group of cases, in which the **osseous symptoms predominate,** and in which the bending of the bones is the most characteristic and striking feature. These are the cases which are likely to come under the charge of the surgeon for deformities of different sorts. Such patients may suffer from time to time from visceral symptoms also, but throughout the course of the disease the bone lesions are those which most arrest your attention. Then there is a third group of cases, characterized by a special tendency to catarrhs. These may be called the **catarrhal cases.** They are children in whom rickets is not very marked, but who tend to have constant attacks of bronchitis or diarrhœa, and in them the rickety element is apt to be overlooked. There is a fourth group which one can profitably recognize—namely, those cases in which there is great **laxity of ligaments** and muscles. I saw yesterday a child who was brought to the hospital with the complaint that he was unable to walk. When a child of three years of age has not yet passed that 'walking milestone' which I spoke of early in these lectures, you may take it that he is suffering from one of three conditions: either he has rickets, or he has mental deficiency, or he is the subject of paralysis. This child I am speaking of was not the subject of paralysis, and there was no reason to suppose him mentally deficient; but he suffered from rickets of the type in which there is extreme laxity of ligaments and great feebleness and flaccidity of the muscles. This is sometimes described as the 'acrobatic' form of rickets, and I shall show you at the end of the lecture some lantern slides which illustrate it.

All these symptoms, no matter which are most pronounced, tend to come on pretty uniformly at a particular period of life, and I want to impress this upon you, because there is nothing more important than to know what diseases are most likely to happen at a particular age. This is a touchstone by which you can invariably recognize a man who has not seen much of practice from the man who has. The beginner is always diagnosing conditions which the more experienced man is aware do not occur at that age. Thus, I constantly find that young infants are sent down from the receiving-room with the statement that they are suffering from rickets. Now, anyone who has seen much of disease in children knows that definite signs of rickets are hardly ever observed below the age of six months; the most likely time for it to come under observation is at about eighteen months of age. By that time the disease usually becomes distinctly marked. It is perfectly true that it *begins* earlier than that. Many people assert that rickets is always diagnosable in the second six months of life ; but even if it were, it is only by the time the child is a year or eighteen months old that the symptoms are likely to impress themselves upon the mother.

We may now pass on to consider the **causes of rickets.** I suppose there is no disease of which it has not been asserted at one time or another that it is due to one of two causes —either to a mysterious nervous disturbance, usually described as trophic, or to the action of bacteria. To this rickets is no exception. There have been some who have asserted that it is due to a trophic influence of the nervous system, and there have been others who have asserted that the whole condition is produced by a micro-organism. I shall ask you to dismiss both these theories from your minds as being, in the present instance, quite unproven. To French authors we owe two other theories : one of them is that rickets is an expression of congenital syphilis, and the other that it is due to dilatation of the stomach. Both of those theories are equally chimerical. The first was put forward by Parrot long ago, and the other by his successor, Comby, at the infants' hospital in Paris. Neither of them has received much support, except from its author or his pupils. And, indeed, I think there are practically only two theories which have to be seriously reckoned with as likely explanations of the production of rickets. One of those is that the disease is due to defective absorption of lime salts, or to the production of lactic acid in the stomach dissolving away the lime salts out of the bones. This one might call the '**lime theory.**' The importance of considering this theory is that a line of treatment has been based upon it. Lime salts have been administered to rickety children under the vague notion that a deficient supply of them lies at the bottom of the production of the trouble. In criticism of this view it may be remarked that, in the first place, there is no reason to suppose that children who suffer from rickets are getting too little lime in their diet. On the contrary, such children are usually being fed on cow's milk, which contains far more lime salts than human milk does, because cow's milk is designed to build up the massive bones of the calf, and is therefore not likely to be deficient in calcium. Further, nobody has yet shown that the administration of lime salts is of any use in rickets. I would also point out that the inorganic matter of the bone, the calcium phosphate, is dissolved away or removed from the bones of rickets as phosphate of lime. Were an overproduction of lactic acid the cause of the mischief, it would be dissolved away as lactate of lime. So the lime theory is falling into discredit, and people are coming more and more to adopt the other theory, which is supported by clinical observation, and also by a certain amount of experimental work—namely, that rickets is due to a **deficiency in the diet of fat and proteins.** And certainly your clinical experience will soon teach you that the main characteristic of the diet which tends to produce rickets is that it is an ill-balanced one, a diet characterized by an excess of carbohydrates and a deficiency in proteins and fat. Whether it is the absence of the fat or of the protein which is the most important factor in producing the disease I cannot tell. One only knows that by increasing the amount of fat in the diet you do much good in rickets, and by increasing the proteins you do good also. It is probably, in most cases, the deficiency of both of these which has produced the disease. But why it is that rickets should result from such a deficiency no one has yet explained. There is no direct relation that anyone can see between a deficiency of fat and protein in the diet and the softening of bones. Yet there is no doubt that animals which are reared upon a diet which is deficient in fat get softening of bones in some way or another.

So much for the theories which have been advanced to account for the production of rickets. I emphasize the last one, because it is by adopting that theory as a working hypothesis that you are certain to be successful in treating your cases. However that theory may stand pathologically, there is no doubt that therapeutics based upon it are justified by results.

Our next point is as to the **diagnosis of rickets.** That is not usually a difficult matter in a straightforward case. You will be struck first of all by the fact that rickety children are slow in their development, slow in passing at least some of those milestones which I drew attention to

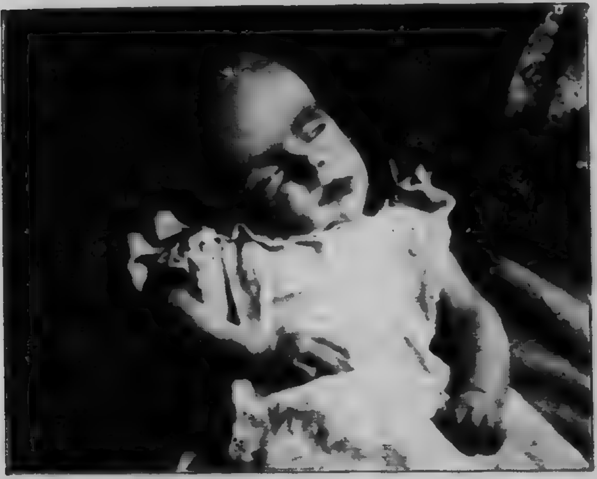

FIG. 13.

SLIGHT DEGREE OF HYDROCEPHALUS, SHOWING GLOBULAR HEAD.

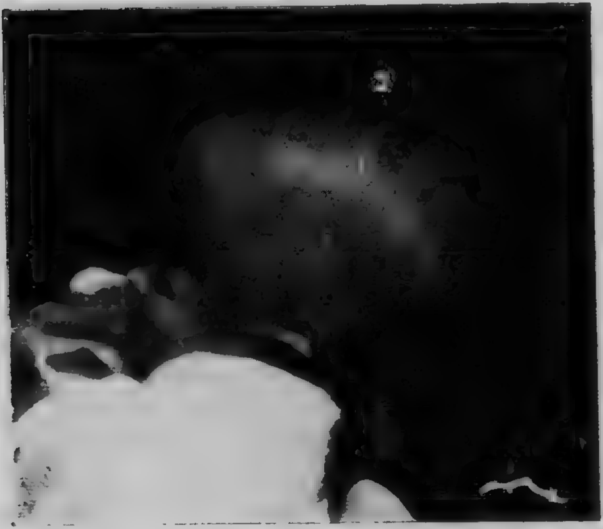

FIG. 14.

HYDROCEPHALUS, SHOWING OUTLINE OF HEAD IN PROFILE. OSSIFICATION WAS HERE VERY IMPERFECT.

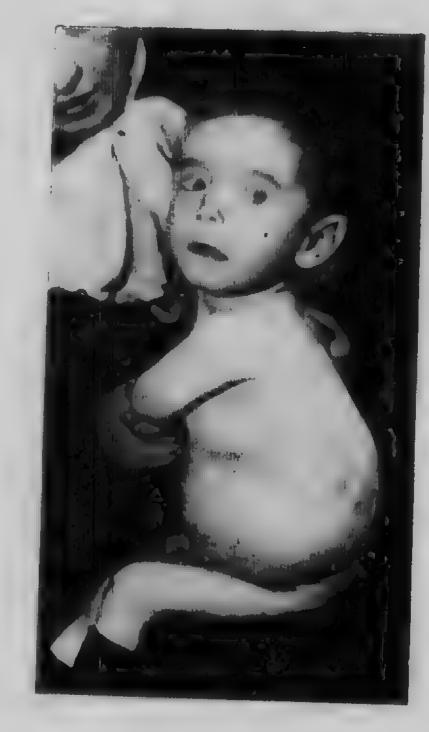

FIG. 15.

RICKETY SPINE, SIMULATING POTT'S CURVATURE.

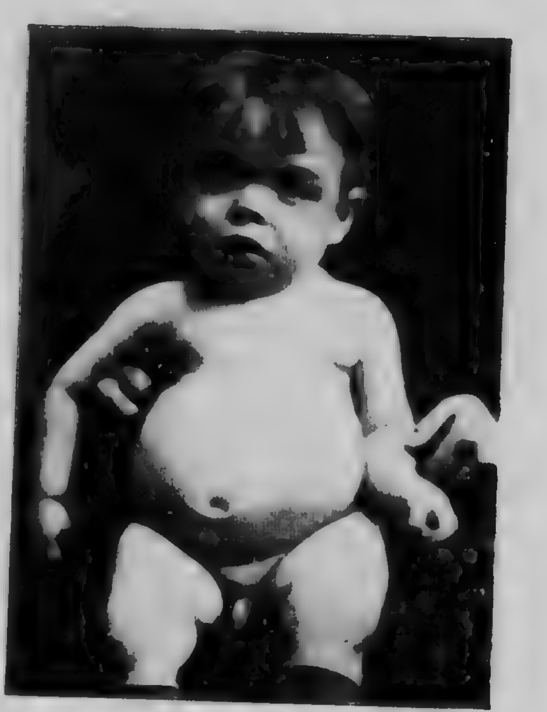

FIG. 16.

RICKETS, SHOWING THE LARGE ABDOMEN, CONSTRICTED CHEST, ETC.

in the first lecture. They get their teeth late, their anterior fontanelle closes late, they sit up unaided late, and they walk late, and all these things are due to the bad development and softening of bones. Then you will recognize, usually without any difficulty, the enlargement of the epiphyses, particularly of the ribs, that being the place where the enlargement comes first. It will also be little marked at the wrists and ankles, perhaps, and here may be some bending of the bones. These are the points to inquire into in the diagnosis of a straightforward case. But there are certain pitfalls in diagnosis to which I want to direct your attention. One of these refers to **the rickety head.** Rickety children will be brought to you under the impression that they are hydrocephalic. That happens many times in practice. How shall you distinguish the rickety child from the child with a hydrocephalic head? The head of rickets has characteristics of its own. It is a long, square head, which looks as if it had been developed in a box. It is not globular, like the head of hydrocephalus, and if you compare the foreheads of the two you will find that in rickets the forehead goes up more or less vertically in front and at the sides, whereas in hydrocephalus it overhangs the eyes and bulges out above the temporal regions; the top of the rachitic head tends to be flat, in hydrocephalus it is convex and globular. By attention to these points you ought not to have any difficulty in distinguishing a rickety head from a hydrocephalic one. There are certain mistakes which are likely in connection with the spine of rickety children, particularly those who belong to the 'acrobatic' group. There is a tendency in such cases towards the production of a prominence in the lower dorsal spine, and they will be brought to you under the impression that they are the subjects of Pott's disease, or tubercular caries. But you will be able to tell a **rickety spine** from a tuberculous one if you remember that one is due to the actual crumbling in of the bone, and therefore cannot be rectified, whereas the other is a mere kinking due to laxity of ligaments, and so can be straightened out. If you hold the child up by the armpits, if the projection is due to kinking it will straighten out,

RICKETS, SHOWING THE LARGE ABDOMEN, CONSTRICTED CHEST, ETC.

RICKETY SPINE, SIMULATING POTT'S CURVATURE.

whereas if it is due to tuberculous disease it will persist. Another pitfall into which you are likely to fall is due to the large development of the abdomen in rickety children. Such cases will be brought to you by mothers under the impression that they are the subjects of tuberculous disease, and many of them are diagnosed as such and labelled with the term 'tabes mesenterica,' against which I have already warned you. There are several reasons why a rickety child tends to develop a **large abdomen.** You know, in the first place, that *all* children tend to have relatively prominent and well-developed abdomens. The reason is that they have a large liver and a small pelvis. In rickets both these factors are exaggerated. The liver of the rachitic child tends to be even larger than in health, mainly from fatty infiltration; and the pelvis at the same time tends to be unusually small and to collapse upon itself, so that there is less room in it for the viscera to sink down into. Moreover, the badly developed and flabby muscles of the rickety child's abdomen allow distension to take place easily; and such distension is rendered more likely because rickety children constantly suffer from gastric and intestinal catarrh, which leads to the development of flatulence. So there are many reasons why a child should suffer from a large abdomen; and if you recognize the rickety element you will be on your guard, and you will not mistake the enlargement for that due to tuberculous disease.

There is still another pitfall into which you are likely to tumble, and that is in attributing to disease of the nervous system symptoms which are really due to rickets. I mentioned such a case a moment ago, that of a child which could not walk when he was three years of age. The diagnosis in such a case, however, should not be difficult. You will find that a rickety child, though he cannot walk, is still able to use his limbs. As he sits in his mother's lap he will kick his legs about; or if you tickle the soles of his feet he will draw up the legs, which, of course, he could not do if they were paralyzed. In rickets the trouble is merely an inability to walk, whereas in paralysis there is inability to move a particular group of muscles in any way at all.

Lastly, I would emphasize the importance of your being able to recognize **the rickety element in many other diseases.** For instance, a child may be brought to you who is mentally deficient, but who suffers from rickets as well; and you can do great good in such a case by treating the rickets, although it is impossible for you to improve the mental condition. Again, when we come to speak of the nervous diseases of children we shall see that rickets is a great predisposing cause in many of the functional nervous disorders, such as convulsions, tetany, and laryngismus; and you cannot hope to treat the nervous disease with success unless you treat the rickets, which is the basis of it. Further, in the case of a child who is the subject of repeated catarrhs, bronchitis, or broncho-pneumonia, or it may be diarrhœa, it may be of the first importance to recognize that these are simply the symptoms of an underlying rickety condition.

That brings us to the consideration of what the **treatment of rickets** should be. Granted the theory of its causation which I tried to bring before you—namely, a faulty diet—your first care must be to alter the child's food. But you cannot hope to do that successfully until you have first put the stomach and intestines into a healthy condition, so that he can digest the food when you give it. So in many cases your first task must be to correct any digestive disturbance which exists. You may have to treat diarrhœa, or gastric catarrh, or want of appetite; and having done that you change the diet. And you will change it in this direction: diminish the amount of starchy things, and increase the amount of proteins and fat. Practically that resolves itself into increasing the proportion of cow's milk and administering cod-liver oil. Of course, if the child can get plenty of cream you can dispense with the cod-liver oil. But always increase the amount of milk. A child of one and a half years should be getting two pints of milk a day, whereas he may perhaps have been getting only half a pint. The yolk of egg is also very useful in these cases, it contains many things which the child needs. It contains phosphorus, fat, and organic compounds of iron, in addition to protein, and I like to give it early in cases of rickets. In very young children it may produce vomiting; but in most cases it is taken quite well.

It should be lightly boiled, so that it is still liquid when given; or you may shake it up with the milk and give it in that way. Many of these cases also do well with the addition of raw-meat juice to their food; or you may even give them underdone meat scraped down. A rickety child must be mainly carnivorous. Rickets might be said to be due to premature vegetarianism, and you have therefore to increase the proportion of animal food, because animal food is characterized by richness in proteins and fat. About the lime salts you need not trouble; there is no occasion to administer them artificially, for milk contains them in abundance. Nor will drugs help you much, unless you call cod-liver oil a drug. If the child is anæmic, give iron in addition. Many people give phosphorus, but I am not convinced that it makes much difference. Attention to the diet and improving the child's hygienic surroundings must be your chief lines of attack, because although it is true that bad diet is the main cause, yet there are co-operating factors. Want of sunlight is considered by many to be one of these. The child should have the benefit of the sun if there is any, and attention should be paid to ventilation, bathing, and clothing. For the deformities you may require to have recourse to surgical aid; but it is surprising how they tend to disappear after a time. Bones which have been very much bent may still, without any particular effort on your part, but simply as the disease passes off, become straightened out again in a surprising way.

In order to prevent the deformities from becoming greater, it is sometimes well to see that the child is kept off his legs. To accomplish this you may require to tie the legs together, or put on light splints which project beyond the foot. And you will require to keep them on for many months, at all events during the day, until the bones have become consolidated again. But in the very severe cases, where the child is five or six years of age, and where there is permanent deformity, surgical means are alone likely to give you much assistance.

Before concluding this lecture I should like to say a word or two about 'late' or 'adolescent' rickets, and about achondroplasia.

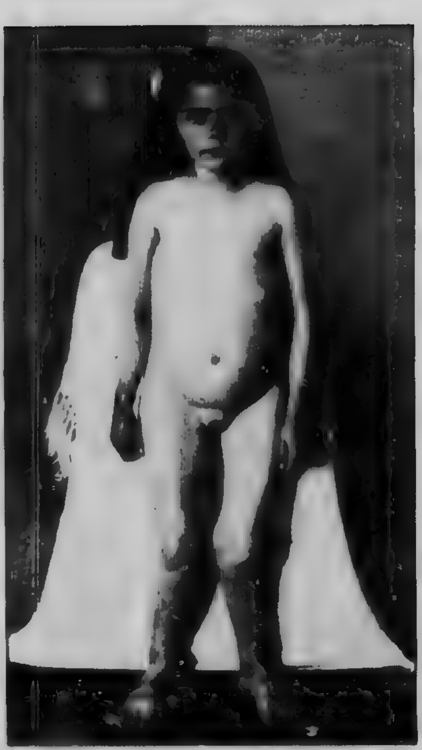

FIG. 17.

LATE RICKETS, SHOWING KNOCK-KNEE AND ENLARGEMENT OF EPIPHYSES.

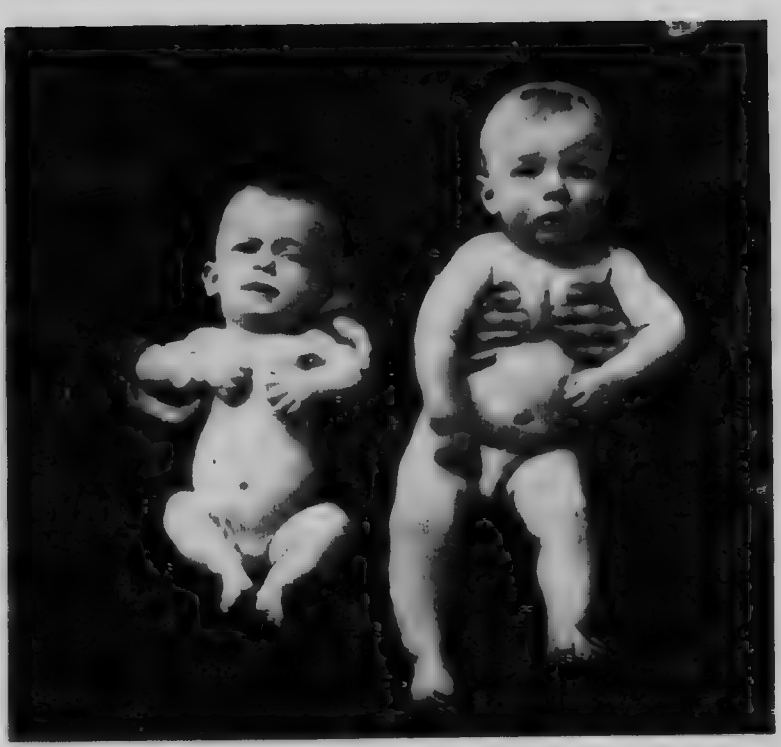

FIG. 18.

TWINS: THE GIRL AN ACHONDROPLASIC, THE BOY NORMAL.

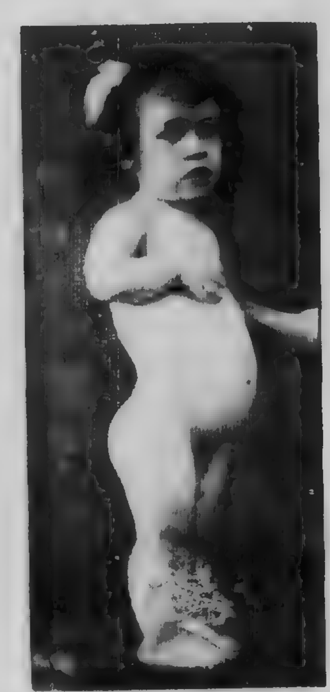

Fig. 19.

ACHONDROPLASIA, SHOWING LORDOSIS.

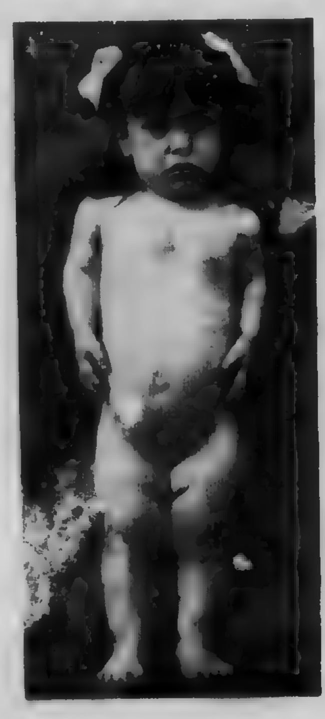

Fig. 19.

ACHONDROPLASIA, SHOWING SHORT LIMBS AND CHARACTERISTIC FACIES.

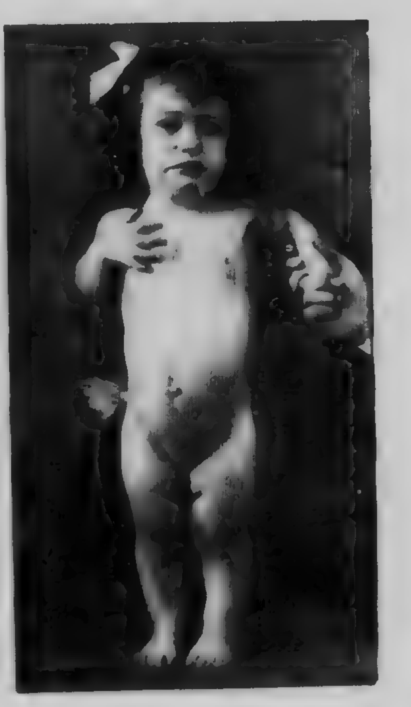

Fig. 19.

ACHONDROPLASIA, SHOWING 'TRIDENT' HAND.

LATE RICKETS.

There can be little doubt that a condition very similar in its effects upon the bones to ordinary rickets sometimes appears in later childhood, or even at the period of adolescence. To this the term 'late rickets' has been applied. Seeing that many of these patients have already suffered from ordinary rickets during infancy, some have concluded that so-called late rickets is merely a continuance or recrudescence of the ordinary infantile form of the disease. This, however, is certainly not true of all the cases, and there seems to be no doubt that late rickets is a genuine disease, although whether it is pathologically identical with ordinary rickets is more uncertain. Clinically late rickets differs from infantile rickets by showing a greater tendency to affect the long bones, the epiphyses of which show notable enlargement, especially, perhaps, about the knees, so that genu valgum and bending of the shafts of the bones are specially apt to result from it. This is very well shown in the photograph I show you, which is that of a girl eleven years of age, in whom the disease had been present for four years. On the other hand, the skull and the viscera do not tend to be affected in late rickets. Skiagrams of the bones in these cases show that ossification is very imperfect.

The treatment of late rickets is almost entirely surgical (by osteotomy, etc.), and I shall therefore not dwell upon it; but, of course, you may need to correct anything that is wrong in the diet or general surroundings of the patient.

ACHONDROPLASIA.

Achondroplasia is a condition which is sometimes mistaken for rickets, although quite unjustifiably, for the two diseases have really nothing to do with each other. Achondroplasia is really a disease of about the third to the sixth month of intra-uterine life, and consists in a partial failure of ossification in the cartilaginous bones. Why this failure of ossification occurs we do not in the least know, but the consequences of it are very striking. A child affected with achondroplasia has a very characteristic appearance (Fig. 19). The limbs are extremely short in proportion to the length of the trunk, producing an appearance which has sometimes been spoken of as a 'human dachshund.' The soft parts of the limbs also hang in folds or creases, like the sleeves of a coat which is too big for the wearer. The hands are short and stumpy, the fingers being approximately of equal length, and, instead of being parallel, radiate out like the spokes of a wheel—the so-called 'trident hand.' As the child grows the normal curves of the long bones become exaggerated, and as the flat bones are unaffected the vault of the skull grows out of proportion to the base, so that the head becomes dome-shaped, with a 'beetling brow,' and a depression at the root of the nose. The face also has a triangular shape, being broad at the forehead, and tapering to a pointed and projecting chin. Lordosis is well marked, and the patient walks with a waddling gait, like that seen in cases of congenital dislocation of the hip. With all these peculiarities the viscera are unaffected, the general health is good, and the intelligence normal. The prognosis is therefore perfectly good as far as life is concerned, which is fortunate, as the condition is entirely insusceptible of treatment.

LECTURE XII

INFANTILE SCURVY

Gentlemen,—Our subject of study in this lecture is that nutritional disease of infancy known as scurvy. This disease used to be called, and is still called by many people, 'scurvy rickets.' That is an unfortunate term, because I think it is now quite well recognized that scurvy in infants has nothing to do with rickets at all, but that the coexistence of rickets in many of the cases is a mere accident, and due to the fact that the kind of diet which is apt to produce scurvy is apt also to produce rickets. Beyond that I believe there is no direct connection between the two diseases, and it is better to use the term 'infantile scurvy' to designate this complaint. The prefix 'infantile' is used because it is apparently not the same disease as scurvy in grown-up persons. It is not the same thing as the scurvy which used to be such a scourge on board merchant ships, or as the 'land scurvy' which occasionally occurs in persons who live in unhealthy surroundings. It seems to be a disease peculiar to infants, and I am glad to be able to say that, just as in the case of rickets, it has been chiefly made known by the labours of English physicians, and particularly, one is proud to say, by men attached to Great Ormond Street Hospital. The first cases were published by Dr. Cheadle in the year 1878, and it was more fully described by Sir Thomas Barlow in the year 1883. His work on the subject was so exhaustive and complete that very little has been added to our knowledge of the disease since that time, and infantile scurvy is now known on the Continent by the name 'Barlow's disease.' Before I pass on to describe it, I should also say that in past years this disease used often to be described under the term 'acute rickets.' When I was dealing with rickets you will remember I told you that there were some cases of that disease which you might call acute, because the symptoms come on rapidly. But that has nothing to do with scurvy, and the term 'acute rickets' should either be abolished altogether, or, if used at all, it should be applied to genuine rickets in which the symptoms come on with unusual rapidity.

What are the **clinical characters** of a case of infantile scurvy? The child will usually be brought to you by the mother with the complaint that he is unable to use one or both of the legs, or that the legs are swelled, or that the child cries when he is touched. These are the commonest things that the mother notices wrong first, and the majority of cases of scurvy which are brought to you will be brought with such complaints as those. When you look at the child you will usually observe that he appears healthy. These children are generally well nourished, they are often of good colour, or, at all events, not conspicuously anæmic; but when the child is being stripped you will notice that he screams a great deal, and is extremely apprehensive. One has been able to recognize cases of scurvy in the out-patient room by the fact that the child starts screaming as soon as he sees the doctor, because he is so afraid he will be handled, for in that way the tenderness of the bones is aggravated. When you come to examine the child more closely you will find perhaps that one of the legs is kept immobile, that it is slightly flexed at the knee, and that the thigh is rotated outwards. On further examination you will probably find signs of rickets—that is to say, slight enlargement of the epiphyses. On opening the mouth you will observe usually that one or two teeth have come through, because infantile scurvy occurs with quite an extraordinary degree of frequency when the first teeth are being cut. Of the cases I have seen in the last two years, twelve or so in all, by far the larger number were children of eight to ten months old. You will commonly find, then, that one or two of the lower incisors are through, and that the gums around them are swollen and of a vivid purple colour. In the upper jaw you may observe that there are

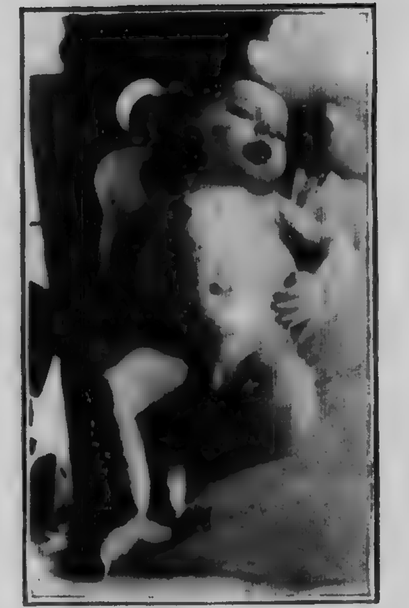

Fig. 20.

SCURVY, SHOWING CHARACTERISTIC POSITION OF LEFT LEG, THE RESULT OF PERIOSTEAL HÆMORRHAGE, AND SOME SPONGINESS OF THE GUMS.

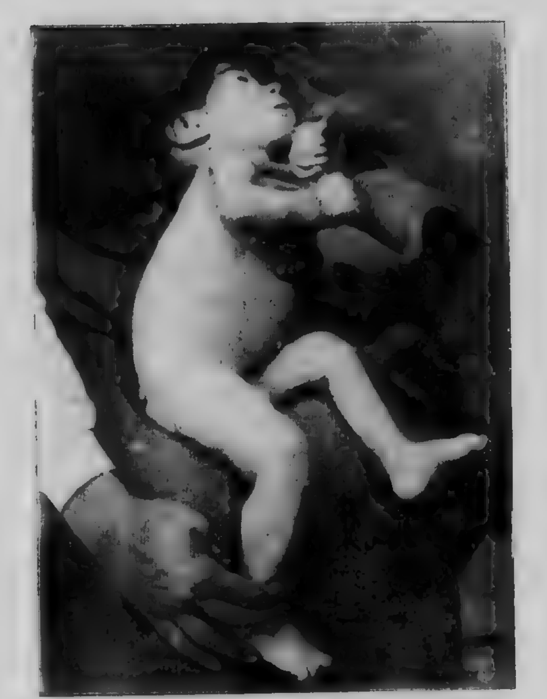

Fig. 21.

SCURVY, SHOWING CHARACTERISTIC SWELLING IN LOWER PART OF RIGHT FEMUR.

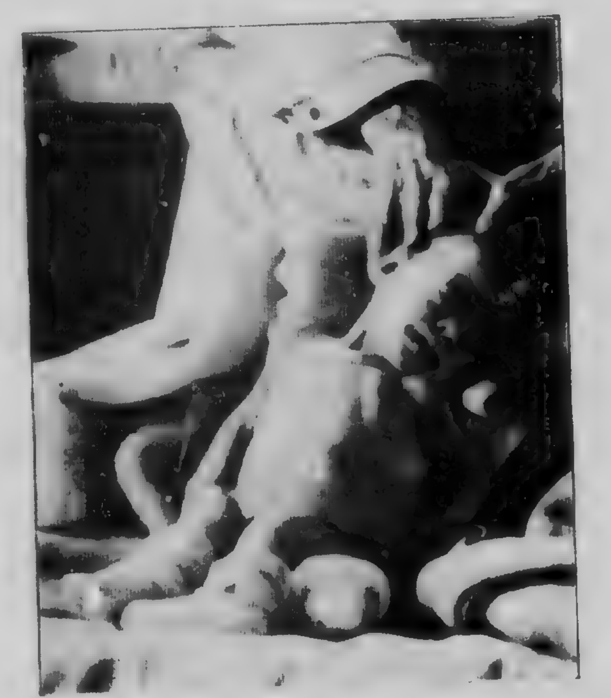

FIG. 22.

ORBITAL HÆMORRHAGES IN SCURVY.

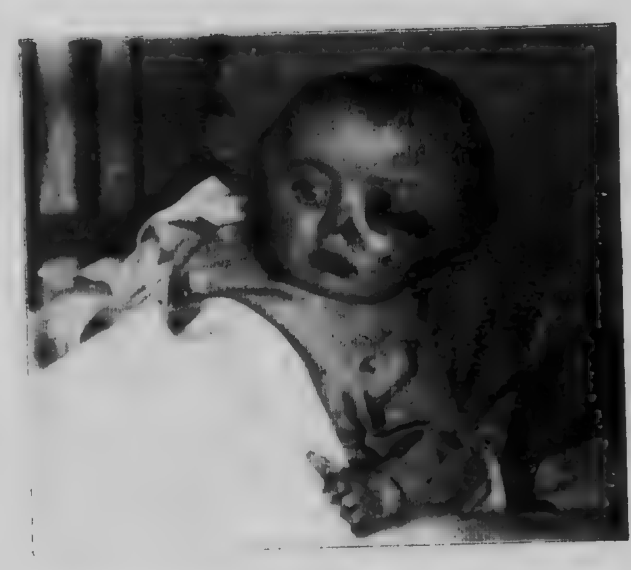

FIG 23.

HÆMORRHAGE INTO ORBIT, THE RESULT OF SARCOMA OF SKULL, SIMULATING SCURVY.

teeth about to be cut, and that the gum over these is in a similarly swelled and discoloured condition. When you come to examine the limbs you will probably find on one or both of the lower extremities, usually towards the end of the femur or the tibia, a thickening which is extremely tender when it is touched, and which presents a sort of boggy feeling on palpation. You may find, further, as minor symptoms of the disease, a tendency to hæmorrhages from the mucous membranes, and particularly the occurrence of hæmaturia. In rare cases the hæmorrhages may take place also into the looser subcutaneous tissues, and particularly into the orbit. These are the clinical signs which you will find when you examine a typical case ; and if you inquire into the history you will find almost invariably that the child has been fed in a particular way—namely, on a patent food, usually along with condensed milk. That is why cases of infantile scurvy are as common, or perhaps even commoner, among the children of the upper classes as in the lower, because these patent foods, being expensive, are to a large extent the perquisite of the rich. It is only extremely rarely that the infant is found to have been fed on the breast. I have never seen such a case myself, but I believe a few are on record in which, in some mysterious way, breast milk has produced scurvy. Once or twice I have seen the disease produced in children who were fed simply on boiled cow's milk, but by far the majority of the cases of the disease have been fed upon a patent food alone or combined with condensed milk.

The pathology or **morbid anatomy** of infantile scurvy is not a difficult matter to explain. It is characterized by a tendency to hæmorrhages, and the seat of those hæmorrhages seems to be determined by the degree of vascularity of the parts. For instance, swelling of the bones is due to hæmorrhage occurring in the deep vascular layer of the periosteum ; the hæmorrhage raises the periosteum, producing the swelling, and the pressure of the effused blood on the nerves produces the tenderness. Sometimes hæmorrhage takes place between the epiphysis and diaphysis, and causes a complete separation of the former, so that it may come to lie quite loose. Very rarely this process of hæmorrhage goes on to the development of suppuration. Similarly, the hæmorrhage round the teeth is due to the fact that the neighbourhood of the teeth is a place which is unusually vascular. That is all one need say about the morbid anatomy. I shall not discuss the chemical pathology of scurvy, as it is still very obscure, but shall pass at once to the question of **diagnosis.**

In the first place, you will have to diagnose infantile scurvy from *rheumatism.* I say that because I have had several cases of infantile scurvy sent to me as rheumatism, in one of which the sufferings of the unfortunate child had been aggravated by the affected limb being painted with iodine. I am certain that this is one of the commonest mistakes made with regard to this disease, that the swelling and tenderness of the bones are regarded as rheumatic in origin. But that is a mistake which you ought never to make, because at the age at which infantile scurvy occurs rheumatism is unknown. Little children below one year of age do not suffer from rheumatism. I do not know why that is, but one knows it to be a fact. So you see this point of differential diagnosis is merely another example of that principle of reasonable probability which I tried to bring before you in a previous lecture—namely, the principle that at certain ages certain diseases are more likely to occur than others. At this particular age rheumatism is not only unlikely to occur, but it does not occur at all.

Another thing which this condition has been mistaken for, and more excusably, is *syphilitic periostitis.* Syphilitic periostitis usually occurs either in children who are much younger than the age at which scurvy occurs, in the form of syphilitic epiphysitis, which I described when dealing with the subject of congenital syphilis, and is a disease seen during the first few weeks of life; or it occurs considerably later in the form of periostitis affecting the long bones, very commonly the tibia, and then the children are distinctly over the age at which infantile scurvy is usually seen. You will find, too, that other signs of congenital syphilis are absent in a case of pure scurvy, and also that in congenital syphilis there is no sponginess of the gums.

Scurvy may also be mistaken for ordinary *epiphysitis,* and that is a fairly excusable mistake which I have known to be made by people who are perfectly familiar with the usual symptoms of both diseases. The only cases in which this error may be made are those in which there is no affection of the gums. But there are other points which will help you. One of these is that the temperature is always raised in epiphysitis, whereas in scurvy it is not usually so to any extent. But one of the best tests of all is to observe the result of treatment. Scurvy properly treated gets well almost at once, so that the therapeutic test is of very great value in diagnosis. If you are in doubt, then, whether a case is one of scurvy or not, put the child upon a diet which is suitable for scurvy and see what happens. If the child has scurvy you will have done right, and improvement will set in at once ; if he has not got scurvy you will not have done any harm. In some cases the therapeutic test may be the only one upon which you can depend.*

There is a fourth condition which affects the limbs, and which it is well to be on the lookout for, so that you do not mistake it for scurvy—namely, *infantile paralysis beginning with hyperæsthesia.* Some cases of infantile paralysis are accompanied at their outset by extreme hyperæsthesia of the limbs, so that whenever they are touched the child cries out. But in such cases the age of the child ought to keep you right. Infantile paralysis is a very badly-named disease, because it is not a disease of infants ; it is a condition which is almost unknown below one year of age. It occurs most commonly between the ages of two and four. In infantile paralysis, too, there will be atrophy or wasting of the limb, and no swelling of it.

The lesions in the mouth and swelling of the gums may be mistaken for other conditions. They may be mistaken, first, for *stomatitis*, and sometimes there will be no means of telling whether it is a case of mere stomatitis or scurvy except by trying the effect of diet. These mouth lesions are much more likely to be mistaken for the ulcerations which occur in connection with leukæmia. I remember seeing a child about six years of age lying in the ward of a hospital, and by its bed was a bottle of lime-juice. The child's breath was exceedingly offensive, and on examining the mouth there was a large excavating ulcer of one tonsil and ulceration and lividity of the gums ; and one was told that the child was suffering from scurvy, and was being treated by lime-juice. But the child did not benefit by the lime-juice treatment. And on examining the blood we found 200,000 white corpuscles, 98 per cent. of which were lymphocytes; in other words, it was a case of acute leukæmia. These latter cases are very often characterized by the occurrence of hæmorrhages into the gums, swelling and sponginess of them, and ulceration. And it is well for you to remember, as I shall have occasion to point out to you when I speak of the anæmias of infancy, that acute leukæmia, although very rare in grown-up persons, is not very uncommon in children ; and it is very often accompanied by those lesions of the mouth. But here, again, the examination of the blood will at once settle the matter.

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* I have lately seen a case of infantile scurvy which had been regarded as one of fractured femur, but the history here should prevent mistake.

Cases which are characterized by hæmorrhages into the orbit may be mistaken for two conditions : for *sarcoma* of the orbit on the one hand, or for that very rare disease called *chloroma*. In the case of sarcoma there will be usually signs of sarcoma elsewhere, and the therapeutic test—change of diet—will be conclusive. The differential diagnosis from chloroma I must defer until I come to speak of it under the anæmias.

Lastly, I would mention that renal hæmorrhage due to scurvy is apt to be mistaken for *renal hæmorrhage* from other causes. I have known a surgeon cut down upon a kidney in such a case, expecting to find a stone, because the child had been suffering from profuse hæmaturia. He found no stone, and the hæmaturia persisted until it occurred to those in charge of the patient to make a change in the diet. They did so, with the result that the hæmaturia disappeared in the course of two days. So hæmaturia from scurvy is a thing which you may overlook. Some people believe that it is a much commoner symptom of scurvy than is generally supposed, and that you will commonly find traces of blood in the urine in a case of scurvy if you look for them. Here, again, I know of no very definite way of making sure of the cause of the hæmaturia except by having recourse to the therapeutic test—changing the diet and seeing what happens.

Before leaving the question of diagnosis, I would remind you that you must be prepared to recognize the earliest beginnings of scurvy before the full-blown symptoms, such as swelling of the limbs, tenderness, and spongy gums, are present. At first the child simply ceases to thrive, his weight no longer rises, he 'goes off his feed,' and is fretful and unhappy. If in such a case the diet be of a kind which is apt to produce scurvy, you should not wait for further developments, but change the feeding immediately. Disappearance of the symptoms will often follow at once.

We may now discuss the **treatment of infantile scurvy.** It was found very early in the investigation of this disease that it could be cured almost at once by insuring the presence in the diet of fresh constituents, such as fruit juice and vegetables. And the routine treatment for you to recommend in a case which comes before you is that the child should be given milk which is raw—that is to say, unboiled. Also, any patent food which the child is taking should be at once stopped. You should give every day a few teaspoonfuls of orange or grape juice, or, if you like, a little raw-meat juice. The child should also be given baked potato. The best way is to bake the potato in its skin, and scrape away the floury part just beneath the skin and shake that up in the milk. You may ask why one should take that particular part and treat it in that way. The reason is that that part of the potato is richest in potash salts. If you put a child who is suffering from scurvy on such a diet, the child will, in the immense majority of cases, get better straight away. There is absolutely nothing in the whole range of therapeutics more striking than the effects which you get from a radical change of diet in infantile scurvy, and there is nothing in regard to which you will deserve or obtain more credit in practice. But, on the other hand, there is nothing in regard to which, if you fail to recognize the condition, you will deservedly get more blame, because it is a disease which it is quite within the compass of medicine absolutely to cure if it be only recognized

LECTURE XIII

THE DYSPEPSIAS OF THE SECOND DENTITION

GENTLEMEN,—In a previous lecture you will remember we considered together the digestive disorders of infancy; and I want now to turn to the study of kindred disorders in older children, and to speak about what one may term 'the dyspepsias incidental to the second dentition.' There are probably many reasons which render children at about the period of second dentition particularly liable to suffer from digestive disorders. One of these, no doubt, is the confinement and want of physical exercise, as well as the mental overstrain, which are incidental to school life, which is usually begun at about that period. You will find that many cases of chronic dyspepsia in children are certainly attributable to that cause. In the second place, owing to the rapidity of growth at this time of life, there is a tendency for the large demand for food to outstrip the digestive power, so that the stomach is no longer equal to the demands made upon it by the tissues. This demand is apt to be met by an excessive consumption of carbohydrate foods, and that, in the third place, is one of the main causes of chronic dyspepsia in children of this age. Coming closer to this subject, one finds a difficulty in classification, just as one did in the case of the dyspepsias of infancy. That difficulty arises, again, from the insufficiency of our pathological knowledge. We do not know what the pathology of many of these cases of dyspepsia is, but I think one may conveniently make the assumption that they are all of them essentially catarrhal in their nature, and I shall speak of them under three headings. First there is acute dyspepsia, or, if you like, acute gastro-intestinal catarrh;

secondly, subacute dyspepsia, or subacute gastro-intestinal catarrh; and, in the third place, chronic dyspepsia, due to chronic gastric and intestinal catarrh.

ACUTE GASTRIC CATARRH.

Acute dyspepsia in older children resembles very closely the similar disease which I have already described in the case of infants. It begins suddenly, as its name implies, and the chief symptom is vomiting. The vomiting is accompanied by prostration, and often by a high temperature, and on examination you find that the tongue is thickly furred, but that otherwise there is very little to be made out. These cases usually arise from one of two exciting causes. One is the consumption of some irritating article of food. What that food is varies in different instances. The stomachs of some children are peculiarly susceptible to irritation by some articles which are eaten with complete impunity by others. Thus, some children have great difficulty in digesting greasy things, and these seem prone in them to produce acute gastro-intestinal catarrh. In other cases the cause may be unripe fruit; but, of course, there are many different kinds of food which may act as exciting causes of the condition. The other great exciting cause which you have to bear in mind is chill. I have repeatedly seen cases of acute gastro-intestinal catarrh brought on in children at about the period of the second dentition by exposure to cold. Thus, I have seen it happen very suddenly after sea-bathing. These cases are apt to begin chiefly with stomach symptoms and to end up with intestinal symptoms; in other words, the catarrhal trouble seems to start in the stomach, and to extend downwards through the stomach into the intestinal canal. That is particularly the case in those instances in which the exciting cause is some error of diet. In them the intestinal trouble ends with diarrhœa, and the diarrhœa, by carrying away the irritating material, leads to a spontaneous or natural cure. The difficulty which you will have in dealing with these cases is in being sure that they are not something else. You may have difficulty in realizing how severe an

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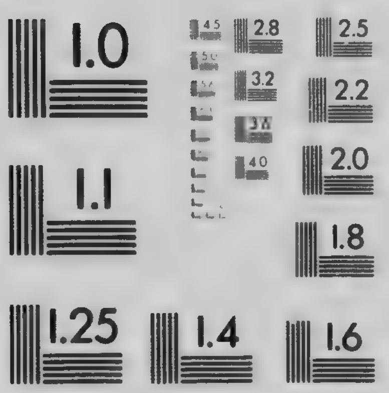

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attack of acute gastro-intestinal catarrh may be ; there may be high temperature, severe prostration, and vomiting, with a furred tongue, and you will, in such a case, be apt to regard the condition as due to something more serious than a mere gastro-intestinal catarrh—for instance, you will think of one of the specific fevers, perhaps of meningitis, or some serious abdominal trouble. I have known a case of that sort in which the abdomen was nearly opened under the impression that the child was suffering from appendicitis.

There is a group of cases in which those attacks recur at intervals, in which a child will go for a few weeks and then have an attack of gastric catarrh, and then go on for another few weeks and have another attack, and the mothers will describe them as **'bilious attacks.'** Some of those cases are probably genuine examples of recurring gastritis, but others are, I think, of nervous origin, and are closely allied to megrim. The term **'cyclical vomiting'** is sometimes applied to them (see p. 361).

The **treatment of acute gastric catarrh** is quite simple. In consequence of the great depression, you should put the child to bed, and withhold all food for a time, or as long as the vomiting is urgent. During that time you may administer sips of iced water in order to allay the thirst. If there is much vomiting, there is no occasion to wash out the stomach, but in cases where nausea is present you may find lavage of the stomach, as in the case of little babies, an important help. Drugs are not of much assistance to you, but the most useful are calomel and bismuth. You should begin at the outset by giving small repeated doses of calomel, ⅙ to ¼ grain every two or three hours until 2 grains have been taken ; then follow that up by bismuth in large doses. Under that treatment, the attack rapidly subsides. And then you have to consider how you are to prevent recurrences of the attacks in future, particularly when you are dealing with a child who is the subject of so-called 'bilious attacks.' How you are to do that we shall consider when I come to speak of chronic dyspepsia.

SUBACUTE GASTRO-INTESTINAL CATARRH.

We may turn next to subacute cases of gastro-intestinal catarrh, a condition which I think is less common than the above, but which may be left behind after the acute attack has passed off, although it may also arise spontaneously and be subacute from the outset. In these cases the fever is not so high as in the acute attacks, but it is more prolonged, lasting perhaps a week or ten days. And as the attacks are accompanied by a tendency to vomit, and sometimes by diarrhœa and a furred tongue, you will readily understand that these cases are very apt to be mistaken for typhoid fever, and many of them undoubtedly used to be written down as '**gastric fever.**' You know that nowadays the term 'gastric fever' has fallen into disuse, and we know that a large number of the cases so described were undoubtedly cases of enteric fever, mild in degree. But I think it is also true that some of them were really instances of subacute gastro-intestinal catarrh. Fortunately, in the agglutination test we have a means of distinguishing those cases from true enteric. Clinically, without that test there is difficulty, because the two diseases simulate one another very closely. So you must be on your guard not to mistake them for enteric, and *vice versâ*. The treatment of them is very much the same as that of acute attacks—namely, rest in bed, restricted diet, and the administration of calomel and bismuth.

MUCOUS DISEASE.

The last group of dyspepsias is the most important of all—that of **chronic cases of gastro-intestinal catarrh.** These cases are extremely common. You will meet them in some form or another in practice almost every day. Together they form a class which is fairly definite, although one can distinguish in it certain special types of the disease. They are sometimes known under the term '**mucous disease,**' that name having been first given to them, so far as I know, by Dr. Eustace Smith, for reasons which we shall see immediately.

I want to describe to you what those cases of mucous disease are like, and to try to draw for you a **clinical picture of the condition.** The child will usually be brought to you by the mother with the complaint that he is wasting. She may tell you in addition that he is languid and peevish, and that he does nothing but 'lie about' all day. Very frequently you will hear the complaint that the child has a cough, and the combination of wasting and cough is often the thing chiefly noticed by the mother, and it is apt to throw you off the scent, and lead you to believe that you are dealing with a case of pulmonary tuberculosis. Over and over again such cases are sent to hospital with the diagnosis of phthisis. When you come to inquire further into the symptoms of the child, you will usually be told that the appetite is very poor. Sometimes, on the other hand, you find quite the opposite condition of things, for there is a voracious appetite. The mother will say that, in spite of the fact that the child eats a great deal, he does not seem to get any fatter. In those cases where the appetite is voracious, you will sometimes get a history that the child has a liking for some quite abnormal things. You may find that he has been eating coal and cinders, or has been going to the wall and picking off pieces of plaster and eating them. To that condition of abnormal appetite the term 'pica' has been applied, and it is not very uncommonly met with among children who suffer from this special form of chronic gastro-intestinal catarrh. When you inquire into the condition of the bowels, you will generally be told that there is constipation. You will hear that the child's motions occur at irregular, and sometimes prolonged, intervals; that the stools consist of more or less hard lumps, surrounded often by material which the mother compares with white jelly—that is to say, by mucus. Very commonly you will get a history that the child is troubled with worms. Less frequently there will be a tendency to diarrhœa, and one characteristic of such diarrhœa which you will often notice is that it tends to come on immediately after the taking of food. The mother will tell you that immediately the child eats a meal there is a tendency for the bowels to act. To that condition the term 'lienteric diarrhœa ' is applied. Proceeding further in your investigation of the case, you will find that these children usually sleep badly at night, that their slumbers are restless and disturbed by unpleasant dreams, and there may be even a tendency to somnambulism. Many of them also have nocturnal incontinence of urine. The mother will frequently tell you also that the child becomes very pale at intervals ; she will say that he goes 'deathly white' for a few minutes, maybe repeatedly throughout the day. These fits of pallor are a little difficult to explain. Some people attribute them to colic. Dr. Angel Money has described them as cases of 'vaso-motor epilepsy,' because he supposed they were due to spasmodic discharges from the vaso-motor centre analogous to the spasmodic discharge from the brain cortex in epilepsy. I need hardly say that that is a purely hypothetical explanation. Certainly they are associated with vascular alterations, perhaps spasm in small arteries or sudden lowering of the blood-pressure ; but what the exciting cause of those alterations in blood-pressure is we do not know.

When you come to inspect the child for yourself, you will usually find that he is at or about the period of second dentition—the majority of the cases occur in children between the ages of five and eight. The child usually looks poorly nourished ; the complexion is pale, and frequently there are dark rings under the eyes. On examining the tongue you will find that it presents certain well-marked features. As a rule, it is covered by a thin fur, through which you can see projecting red points, which are the enlarged fungiform papillæ. Or, in other cases, and these perhaps the most typical, the tongue has a glazed appearance, and is of a slightly yellowish or fawn-coloured tint, looking, as it has been described, as if it had been brushed over with a solution of gum. In yet a third group you will find that the tongue shows peculiar mapped-out areas, curious irregular patches where the epithelium seems to be denuded, which have been fancifully compared to the continents in a map of the world ; hence it is called the 'geographical tongue.' On proceeding to examine the child further, you will find that the skin is dry and harsh. Usually you cannot make out any physical signs of disease in any of the great viscera ; the heart, lungs, and abdominal organs appear to be healthy. But when you come to inspect the throat, you will almost invariably find that it is in a more or less unhealthy condition, that there is chronic pharyngitis, more or less enlargement of the tonsils, and very frequently adenoids in the naso-pharynx. It is really to the unhealthy condition of throat that the cough in these cases is due, and that fact I cannot too strongly impress upon you. The majority of cases of cough which are brought before you in children are cases of throat-cough, and not lung-cough at all. When you examine the urine you will often find that it is turbid, owing to a deposit of urates. Sometimes in such a case the mother will describe the urine as being 'milky' when passed, that appearance being due to pale urates. On chemical examination you will often find that it contains albumin, probably in many cases nucleo-albumin. Such children make up a large proportion of the so-called 'cyclic albuminurias,' which are, I think, better termed simply functional or postural albuminuria—that is to say, cases in which there is no organic disease of the kidney, but in which albumin or protein appears in the urine at particular times of the day, usually after the child has got out of bed and is going about. The morning urine which has been secreted in the night is usually free from any trace of albumin at all.

Such are the chief symptoms of a typical case of this so-called mucous disease. But you will frequently meet with cases which are not so typical as that, but in which one or other group of symptoms tends to overshadow the rest. And I think you can distinguish several such special groups. First, you can distinguish those in which the **constitutional symptoms predominate.** By that I mean that the chief symptom is the wasting, accompanied by mental depression and languor, and perhaps by some degree of mental irritability. Then there is a group in which the **stomach symptoms predominate.** The chief complaint in those cases is that there is pain after food—a pain which you will frequently have difficulty in referring to the stomach or colon, but which may probably have its seat in both, owing to flatulent distension of the stomach and large bowel. Other cases which certainly belong to this kind of intestinal disorder will be brought to you simply for **worms,** and I want to impress upon you at this point that you should always regard worms as a symptom, and not as a disease. They are a symptom of chronic intestinal catarrh, and not a disease in themselves. Other cases will be brought to you solely with the complaint of **lienteric diarrhœa**—that is to say, the diarrhœa is so prominent a symptom as to overshadow all the rest. And, lastly, you will have cases brought to you for **restless and disturbed nights,** the so-called pavor nocturnus, or night terrors.

We will pass on now to glance at the **pathology** of this disease so far as we know it. It is difficult to speak at all dogmatically in this matter, because there is an almost complete absence of post-mortem evidence in regard to it. It is not a fatal disease ; it leads to much ill-health, and it predisposes the child to suffer from more serious disorders, such as tuberculosis, but it is not in itself inimical to life, and thus there is no opportunity for investigating its morbid anatomy. Dr. Eustace Smith has a very neat theory to explain all the symptoms detailed above. He says that mucous disease is due to an over-secretion of mucus throughout the whole alimentary canal ; that the excess of mucus produces in the stomach sluggish and painful digestion ; in the intestine it wraps round the intestinal contents, the consequence of which is that the digestive juices get imperfect access to the food, and hence absorption is interfered with, and as a result the child wastes. Moreover, the scybalous masses in the large intestine get coated over with mucus, so that the wall of the gut gets no purchase over them to squeeze them along, but glides over the masses ; hence the frequency of constipation. In addition, the mucus forms a good nidus for the worms to live and feed and lay eggs in. One might go further, and say that in the cases in which there is albumin in the urine it is very often a nucleo-albumin, and that this is due to analogous over-secretion from the cells lining the urinary passages. And one might also point to the throat, and say that there, too, one finds chronic catarrh and the production of an excessive quantity of mucus. This theory, then, as you will see, is very neat and ingenious, and it does explain fairly satisfactorily most, at least, of the symptoms. But it is one which it is very difficult to be sure about. In those cases in which you get the passage of masses of mucus from the bowel there is a considerable amount of evidence in support of it. But this does not always occur, and the real importance of the theory is that the treatment which is based upon it is certainly justified by its results. That treatment I shall describe to you immediately. There are many who do not accept the mucus theory, but [illegible] consider that these cases are more obscure than any such explanation as that would lead you to believe. They think one is here dealing with a disorder of metabolism—a defective power of assimilation which they compare to the gouty condition in grown-up people. They say—and especially French authors—that those children have the 'arthritic diathesis,' whatever exactly that may mean. There are physicians in this country who also maintain that view, and believe that these cases are due in some way, not completely understood, to an imperfect elaboration of protein material, which leads to the production of an excess of uric acid, pretty much as you know takes place in gout. Between these two theories I shall not attempt to decide, because, as I have said, there is no definite ground to go upon in the argument. I want further to point out to you that whatever the true theory of the mode of production of the symptoms is, there can be no doubt whatever about one frequent exciting cause of the disorder, and that is a matter which it is of more importance for you to know. It is the excessive consumption of sugar. That is so common and so well recognized a cause of this disease that you might almost call it the dyspepsia of sweet-eating children. You will remember that cane-sugar is eminently a producer of mucous secretion. If you take cane-sugar into the mouth and suck it, the mouth becomes sticky, not merely from the presence of the sugar, but from the secretion of a viscid mucus by the salivary glands. So that this fact would also fit in quite well with Dr. Eustace Smith's theory.

We come now to the **diagnosis** of chronic gastro-intestinal catarrh. I have said that these cases will come before you with great frequency, and there is one condition which you are particularly apt to mistake them for, and that is tuberculosis in some form. One reason for this is that the child is so often brought for cough and wasting, a combination of symptoms which in a grown-up person you are apt to associate with the presence of pulmonary tuberculosis. But I would ask you to remember once and for all that pulmonary phthisis is a rare disease in children. Tuberculosis at this age does not tend to take the pulmonary form. If you are in doubt whether you are dealing with a case of latent tuberculosis somewhere, I advise you to give heed to the temperature ; make a point of taking it night and morning for a few days. Dyspeptic cases are not accompanied by fever, or, at all events, the fever is not continuous. There may be transitory attacks of fever during the exacerbations of the condition. but these only last a short time, whereas cases of tuberculosis, no matter where situated, tend to be accompanied by nocturnal rises of temperature, which persist for several weeks.

Finally we arrive at **treatment.** If you set about this in the right way, I think I can promise you very satisfactory results. But if you are not familiar with these cases, you will find them very troublesome and chronic, and difficult to cure. Seeing that I have just mentioned that the excessive consumption of carbohydrate food, particularly sugar, is undoubtedly one of the chief exciting causes of this form of dyspepsia, the main line of treatment must be dietetic. The first thing to do is to attend to the food. The sheet I have put into your hands describes to you the chief lines upon which the **diet** for such cases should proceed.

Directions for Diet in Chronic Dyspepsia.

No food must be taken between the regular meals.

No sugar or sweets of any sort are to be eaten.

The child must *not* eat: New bread, potatoes, peas or beans, turnips or carrots, pickles, pastry, jam, syrup or treacle, or cakes.

The diet should chiefly consist of: Stale bread or dry toast, with butter or dripping, bacon, eggs, fish, meat, milk, plain milk pudding (except cornflour, arrowroot, or sago), and green vegetables in small quantity.

You will see that it consists essentially in the limitation of carbohydrates, and in the almost total prohibition of sugar. If you will keep that broad principle before your minds, you will have no difficulty in adjusting the details. Next to a change of diet I put in order of importance **change of air.** Repeatedly one has seen cases which were hanging fire and not doing well get better right away when they were sent to a convalescent home at the seaside. That is probably to be explained on the grounds which I have already mentioned, that many of these cases are due to a lack of physical exercise and confinement to school for long hours, and to unhealthy homes. Next we come to consider the question of **drugs.** You will find that the drugs chiefly of use at the outset of the case are alkalies and aperients. I think aperients are useful no matter what the state of the bowels—whether there is constipation or not—and it is a good routine plan to begin by using them. The usefulness of those remedies may also be accounted for on the excessive-production-of-mucus theory. According to the holders of that theory, one should administer alkalies because they are an admirable solvent of mucus. And one administers aperients to get rid of the mucus which has been so dissolved. Whether the explanation is right or not, at all events you will get great success from the adoption of this line of treatment. I use some such prescription as this : Pot. bicarb., pot. cit., āā 5 grains ; tinct. nucis vom., 1 minim ; compound infusion of gentian, 2 drachms, given with a little water a quarter of an hour before the meals. The action of each ingredient of this prescription is believed to be this : The bicarbonate of potash dissolves the mucus. The citrate is given largely because these children tend so often to have thick urine and the passage of urates. The nux vomica is given to increase appetite, which is usually bad. The gentian is given for the same reason, and also because it has a mild astringent action on the mucous membrane of the stomach. In cases in which nervous symptoms predominate bromide of potash should be added to the prescription.

The **aperients** I like to begin with consist of rhubarb and mercury in the following combination : Rhubarb powder, 8 grains ; bicarbonate of soda, 10 grains ; gray powder, 1 to 2 grains, according to the child's age. This should be given every night, or in a milder case every other night. Rhubarb is selected because it has not only an aperient action, but it has an astringent after-action, and a tonic effect upon the flabby mucous membrane. One adds bicarbonate of soda for the same reason as in the other mixture; and many cases are undoubtedly greatly benefited by mercury in some form or another, probably because mercury seems to have a peculiar specific influence in diminishing catarrh of the alimentary canal. After a course of such treatment for a fortnight or so, you will probably find a striking degree of improvement; the child's appetite will have returned; he will begin to gain flesh; he will no longer suffer from restless nights, and you will have reached a stage at which you can begin the admistration of tonics, which must include iron in some form or other. A very good form is the wine of iron, given with an aperient in the form of aloes.* I want particularly to impress upon you that you should defer the administration of these tonics, particularly iron, until you have got the tongue clean and the stomach in a healthy condition. There is no commoner mistake than to treat these cases at once with cod-liver oil and Parrish's food as soon as they are seen. The doctor is apt to order them iron to make them strong and cod-liver oil to make them fat; and such treatment, if begun too early, does harm instead of good. It is a good rule in medicine not to begin the administration of iron until the stomach is in a healthy condition, because iron is almost always an irritant to the stomach, and it is not absorbed and made use of until the alimentary canal is brought into a condition somewhat approaching that of health. On the other hand, malt extract is undoubtedly of use sometimes in promoting the assimilation of starch foods. Although that should be your general line of treatment, you will find that special symptoms in particular cases may call for special remedies. Many patients, for instance, are troubled with gastric pain or colic, which may be extremely obstinate, and sometimes the only thing to which it will yield is opium. The best form in which to give it is Dover's powder, and 2 or 3 grains ought to be sufficient. If you have lienteric diarrhœa to deal with, you will find the best thing to do is to give small doses of opium before meals—perhaps 2 minims of the tincture of opium may be enough—and it is well to combine with that a drop of Fowler's solution and a little nux vomica. Lienteric diarrhœa is brought about by peristalsis in the upper part of the bowel being too active and too easily excited reflexly, so that when food is introduced into the stomach, instead of merely exciting the stomach it starts a wave of peristalsis, which spreads through the intestine and leads to a motion. Opium is often the only thing which will allay that and prevent the diarrhœa. Should headache be a prominent symptom, as it is apt to be, I recommend you to pay particular attention to the refraction of the eyes. You will often find that there is a slight error of refraction, astigmatism, or hypermetropia, and if you correct that by means of suitable spectacles the headache disappears at once. The cough, which is so common in these children, may necessitate local treatment directed to the throat; you may require to use nasal douches, or washing out of the back of the throat with an alkaline and antiseptic solution. But in many instances you have to proceed sooner or later to the removal of the tonsils, and perhaps also of adenoids. But the relation of adenoids to these cases I shall discuss more fully in another lecture.

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* Vinum ferri, ʒi.; decoct. aloes co., ʒi. or ʒii.—after each meal.

I have already mentioned that children who suffer from this form of indigestion are very prone to have worms, and I may conclude what I have to say to-day by a brief discussion of this complication.

INTESTINAL WORMS.

There are three varieties of worms which may inhabit the alimentary canal of children. There are the tape-worm, the round-worm, and the thread-worm. On rare occasions other forms are met with, but for practical purposes these are the only worms we need discuss as affecting children in this country.

TAPE-WORMS.

Two varieties of tape-worm are found in children. These are the *Tænia cucumerina* and the *Tænia mediocanellata.* The first of these is much rarer than the second, and is, in fact, not very well known. It is a comparatively short tape-worm, and its presence is recognized by the passage of small oval segments. Both the cases I have hitherto met with were in infants at the breast, as is usual with this worm; this extraordinary point in the distribution of *Tænia cucumerina* naturally causes difficulty in diagnosis. You may ask, How in the world does a child nursed at the breast get a tape-worm? It appears that this particular worm is always acquired from cats, and in every case it is found that the sufferer has been in close association with one of these animals.

Tænia mediocanellata is the common tape-worm with which you are all familiar, both in children and in adults. It seldom occurs in young children, but is frequent at and after the second dentition.

Children are often brought by their mothers for advice with regard to worms. Various symptoms are supposed to be caused by their presence, such as wasting in spite of good appetite, abdominal pain, and so on. Let me say at once that, except possibly in the case of thread-worms, I know of no symptom that points definitely to the presence of worms. The only method by which you can diagnose the existence of worms in the alimentary canal is by actually seeing segments or entire individuals in the fæces.

Treatment of Tape-Worms.

This is not such an easy matter as one might suppose. You will often fail to get away the whole worm, and this is in many cases because the condition is not taken seriously enough, and treatment is half-hearted.

The patient should remain at least twenty-four hours in bed, and for some hours before the anthelmintic is given, and until the worm is passed no solid food at all must be allowed. Begin in the evening by giving a purge so as to clear the alimentary canal as much as possible, and thus expose the worm to the direct action of drugs. A full dose of castor oil, or of liquorice powder, or of calomel, should be administered; it does not very much matter which, provided the dose is a sufficient one.

Of the anthelmintics none surpasses male fern, which, on account of its nauseous taste, is best given in capsule in 15-grain doses every quarter of an hour for four doses. A child of the age at which tape-worm is common will stand such a dose quite well: children, in fact, bear all aperients well in comparison with adults. Having given four such capsules it is best to wait for an hour or so, and then to give another purge to clear out the worm. As a quickly-acting aperient is wanted, an effervescing saline, followed by a hot drink, is useful.

It is very important to attend to details in the passage of the resulting motion. It should be passed into warm water to diminish the risk of breaking the worm. There should be a sheet of black crêpe on the bottom of the vessel, which assists the search for the head of the worm; this can often, with a little practice, be recognized by the naked eye. It is only when the worm is completely evacuated and the head seen that one can be sure of success.

If the treatment fails it is not advisable to repeat it until after an interval to allow of growth. As a rule this takes about two months. The essentials are rest, starvation, purgation, full doses of an anthelmintic, and care in the way in which the worm is received when passed.

About the treatment of *Tænia cucumerina* I cannot tell you much. Personally I should be rather afraid of giving male fern to a little baby; I should prefer calomel, and perhaps small doses of santonin as well. Both the cases to which I have referred had calomel only, and soon ceased to pass segments, though I had not the opportunity of searching for the head.

ROUND-WORMS.

As in the case of the tape-worms, there are no symptoms characteristic of this worm, the *Ascaris lumbricoides*. There is, however, no doubt that it may cause reflex nervous disturbances of various kinds. I have myself seen cases of convulsions which ceased after the passage of a round-worm. These worms are probably derived from certain kinds of food, particularly raw vegetables, such as salads. I once had to do with a family of strict vegetarians, in which the father, mother, and both children were suffering from round-worms, and I do not doubt that it was from their vegetables that they had contracted the parasites. With a view to prevention it is well to see that anyone who has had a round-worm should avoid such foods, or at least take the most careful precautions about washing and cleaning them.

The round-worm is the only kind of common worm to which any danger attaches. The danger arises from this reason, that they are always prone to wander. When the worm wanders upwards into the stomach, it is generally promptly vomited ; but it may obtain entrance into the larynx and cause suffocation, or into a bronchus and set up bronchiectasis. If a child passes a round-worm it should be at once put through a course of treatment, for these animals often hunt in couples. Fortunately round-worms are more easily got rid of than tape-worms.

Santonin is the best remedy, for it is effective and safe ; amongst numerous other anthelmintics, thymol has been a good deal used. Santonin should be exhibited as a powder in this combination :

Santonin	gr. ⅙
Calomel	gr. ⅙
Sugar	q.s.

Give one such dose for every year of the child's age every night for three or four nights. So much, then, for round-worms : their treatment is simple, and when once the worm is seen there is no anxiety as to any of it having been left behind.

THREAD-WORMS.

The third and commonest variety of worm that attacks children is the *Oxyuris vermicularis*. It is very important to realize that thread-worms are not a disease in themselves, but a symptom of an unhealthy state of the large bowel. I doubt very much whether a thread-worm can exist in a bowel that is healthy. The sort of condition required is one of catarrh associated with the presence of mucus. It is in the folds of the swollen mucous membrane that the worms lodge, and to be successful in treatment this condition must be remedied.

Another difficulty is that the patient, if a child, is constantly reinfecting himself. Further, the ova are often deposited in the vermiform appendix, which forms a regular breeding-place for oxyuris. When once one has seen such an appendix full of young worms, it can be realized how difficult it is to stop the constant reinfection.

This brings us to the symptoms caused by thread-worms. There are certain purely local effects, such as irritation, scratching, and so on. Beyond these, it is quite possible that incontinence is sometimes due to worms, but there is much less ground for the belief common amongst mothers that picking at the nose is evidence of them. As regards pallor, abdominal pain, wasting, headache, and other vague symptoms, I am convinced that these are really due to the state of ill-health which permits the worms to flourish, and which I described in the earlier part of this lecture.

To get rid of the worms it is necessary to deal with this underlying condition by the methods detailed above.

Some authorities recommend santonin by the mouth for thread-worms. I am not at all sure that it has any anthelmintic effect on them, and if you adopt the methods I have indicated there will be no need to use it.

To deal with the worms locally, various injections have been proposed. Infusion of quassia is an old-fashioned prescription, and a very good one ; solution of common salt, in the proportion of an ounce to the pint, is another ; and infusion of garlic is sometimes very effective. These may be safely used every night for a time. The following may be injected every other night for three doses with good results :

Oil of turpentine	ʒii.
Santonin	gr. ii.
Starch mucilage	℥vi.

There are two different systems of giving an injection for these worms. Some physicians advise a large injection so as to reach as far up the bowel as possible. Others prefer a small one that it may be retained as long as possible. My own feeling is in favour of small injections, which have the advantage that they cause much less discomfort, as the process is one which is frequently repeated ; this is an important point when dealing with young children.

The injection should be given warm, and quite slowly, through a funnel. The pelvis of the patient should be raised. An injection of 6 ounces when given thus may in a child reach the transverse colon by reversed peristalsis. This small quantity is safer and less likely to be painful than the larger doses ; in addition, it is far less likely to damage the intestine, whose wall is thinner and more easily ruptured than is often thought.

A small portion of ung. hydrarg. inserted into the rectum every night for a few nights is also a very useful local remedy.

It is well to order a little weak white precipitate or nitrate of mercury ointment for local application, to relieve itching and to prevent wandering of the worms.

I will only repeat that local measures are less important than attention to the state of the intestine, for if the worms are only killed off locally they will certainly come back, a process which has been known to continue for years.

If I have mentioned to you but a few lines of treatment it is because I believe that if they are thoroughly carried out satisfactory results will always be obtained.

LECTURE XIV

RHEUMATISM IN CHILDHOOD

GENTLEMEN,—I wish to bring before you in this lecture the subject of rheumatism as it manifests itself in early life. Now, there are few diseases commoner than rheumatism in one or other of its many forms in childhood, and one may say also that there are few more serious, or in relation to which your responsibility is greater; for rheumatism, you will remember, in early life is often the parent of cardiac disease in later life, and many people suffer from disease of the heart for a large part of their adult existence because they had an attack of rheumatism which was overlooked by the medical attendant in their childhood. That is why I want to impress upon you your responsibility in relation to this disease. It is a condition which, if properly treated, may be cut short, and, if cut short, many consequences which would otherwise blight the life and activity of the subject of it may be prevented.

By rheumatism in children I mean something quite specific and definite. You know that the term in relation to adults is one which is used in a very vague sense. One talks of 'chronic rheumatism' of a joint, for instance, without having any very clear idea before one's mind of the exact pathological state of that joint. Now, in the case of children one does not talk in that loose way. One means by rheumatism **an acute specific disease,** which is almost certainly due to a specific micro-organism. Indeed, I think the modern view that acute rheumatism is due to a micro-organism receives strong support from the study of the disease in childhood. All micro-organismal diseases, all acute specific fevers, as you know, tend to be commoner in early life, and the great frequency of rheumatism in childhood is an *a priori* argument in favour of the view that it also is due to a specific micro-organism. Most people have now come round to that opinion, and they regard rheumatism as a morbid entity, as definite and precise in its pathology as typhoid fever or diphtheria. Now, though rheumatism may certainly be regarded nowadays as being due to a specific organism, you require for its production not merely the organism, but a suitably prepared soil. Clinical observation shows that all children are not equally subject to attacks of rheumatism. There are few diseases, indeed, in which heredity plays a more definite part You constantly find, when you inquire into the family history of children who are suffering from one or other manifestation of acute rheumatism, that other members of their families have been similarly affected ; and I think you can recognize when you meet them children who are so predisposed by heredity. I have often pointed out to some of you in the wards and in the out-patient room the **typical rheumatic child**, from whose facies you can say that he is prone to become the subject of this disease. These children are usually past the second dentition, for young children rarely suffer from rheumatism. They are dark rather than fair ; their hair is dark, the eyes are dark, and they have long, dark eyelashes. At the same time, they have a peculiarly white skin and a very good complexion ; they have a clear, bluish-white sclerotic, and they have often very well-formed, massive teeth, and particularly large, square, central upper incisors. They also exhibit very constantly what is termed a neurotic temperament—that is to say, more than other children they are subject to minor nervous disorders. I think you will find it an advantage to have such a picture of the typical rheumatic child before you. It has occurred to me several times by recognizing these characters to suspect rheumatism, or to be specially on lookout for it, or to be able to say of a particular child that he would easily become the subject of rheumatism, and so to advise the taking of special precautions against infection.

Now, unfortunately, we do not know how those children become infected. We do not know definitely the path by which the rheumatic organism gains access to the body. We are unable to trace it as we can trace, for instance, the path of the tubercle bacillus. But I think there is at least a reasonable amount of evidence to show that infection takes place in perhaps the majority of cases through the throat. Now, **unhealthy throats** are so common in children that one need never be surprised that the local resistance there is lowered; and I believe that a great many diseases could be prevented—and among them rheumatism—by a greater attention to the state of children's throats. I shall have occasion, however, when I come to talk of adenoids in infancy and in childhood, to speak a little more fully upon this subject. The organism, having gained access by the throat or by some other route of which we are ignorant, tends to manifest itself in certain tissues in children more than in others, and in particular the fibrous tissues and membranes are subject to its attack; I mean such membranes as the valves of the heart, the tendons, the sheaths of muscles, and the fibrous aponeuroses. These are the **tissues which are most vulnerable,** it would appear, to the attacks of the rheumatic bacillus. Now, in this respect rheumatism, as it shows itself in children, differs considerably from the same disease as you see it in grown-up people, and it is much to be regretted that the ordinary text-book description of rheumatism is drawn from a study of the disease as it occurs in adults. Rheumatism ought to be described as it occurs in children, because it is in children that you see it in by far its most virulent manifestation. No one who has seen much of the disease in children will fail to recognize that the joint manifestations are often very trivial or altogether absent, so that, had it been studied primarily in children, it would never have been called acute articular rheumatism, which is the name usually given to it in text-books. That title is correct as regards grown-up people—in them the joint manifestations dominate the whole picture—but in children it is not so. In them rheumatism may invade the body, and almost pass over the joints altogether; or, at all events, the joint affection may be so trivial and transitory that it may be completely overlooked. On the other hand, it is an unfortunate fact that it is the valves of the heart more, perhaps, than any other part which tend to be the chief point of attack of the rheumatic poison in children, and for that reason the disease assumes in them a degree of danger and importance which is wanting in the case of the adult. Not only is the joint affection often exceedingly trivial in children, but you will often find that the degree of fever is extremely slight. Children suffering from acute rheumatism may have a rise of temperature amounting to only one or two degrees, and they hardly ever exhibit that dangerous rise of temperature which is spoken of as rheumatic hyperpyrexia. For some reason the whole febrile side of the disease is much less marked in the young. Indeed, some people go the length of saying that it would be more true to describe the endocarditis as the essential part of acute rheumatism in children, and the joint manifestations as mere complications, and that it is quite wrong to assume the attitude which one is apt to do, and speak of the joint lesion as the essential thing and the endocarditis as the complication. The next point I want to emphasize is that the manifestations of rheumatism in early life are far more protean than in grown-up people There are all sorts of forms of rheumatism in children which you may be apt to think are not rheumatic at all unless the fact is first pointed out to you. One may classify the **chief manifestations** of the disease as follows : First, articular inflammation, the ordinary synovitis of joints ; secondly, muscular or fascial rheumatism ; thirdly, rheumatic nodules ; fourthly, endocarditis, myocarditis, and pericarditis ; fifthly, erythematous eruptions of various sorts ; sixthly, chorea ; seventhly, pleurisy ; and, lastly, tonsillitis. I have not mentioned these rheumatic manifestations in the order of their importance. If one had done this, one would have placed the heart manifestations first. I have merely put them in series to show you how varied are the forms assumed by rheumatism in early life. Now, these different manifestations of the disease may coexist, or they may succeed each other in any order. Given a child predisposed to rheumatism, he may show at or time a few articular pains.

These pains may pass off, and a few months later the child may have a little muscular and fascial rheumatism somewhere; later on, perhaps, he gets an attack of chorea, and that may be complicated by an eruption of rheumatic nodules and by some inflammation of the pericardium; or the child may have repeated attacks of tonsillitis, and occasionally fugitive erythematous eruptions, or even, perhaps, an attack of pleurisy; and in that way all these events may be spread out over childhood, one occurring at one time and one at another, but all being simply different manifestations of the one poison—namely, that of acute rheumatism.

I now want to look briefly with you at each of these members of the rheumatic series.

The **joint pains** I have already referred to. I have pointed out that they are often so slight as to be overlooked. But if you watch your cases of rheumatism carefully you will usually find *some* involvement of the joint at least at one or other period of the illness. It is very rare for a case to run anything like a prolonged course without there being at any rate some articular inflammation, but its degree may be extremely slight. Further, the inflammation of the joints is not so intense in its local manifestations as in the grown-up person; there is much less swelling and heat, hardly ever any redness, and there is comparatively little pain.

The **muscular and fascial rheumatism** of childhood deserves your special attention, not because it is of importance in itself, but because it should act to you as a danger-signal. The pains of muscular and fascial rheumatism are commonly called, in the nursery, 'growing pains.' That, gentlemen, is a phrase which has crippled a great many hearts. Mothers and nurses are apt to regard growing pains as something physiological, something to be expected in growth. I need hardly say to you that that is an absolutely mistaken view. Those so-called growing pains are, in the great majority of cases, nothing more than the local manifestations of rheumatism in the muscles and fascia, and if they are neglected and the child is allowed to go about with them, he may very easily slide into endo-

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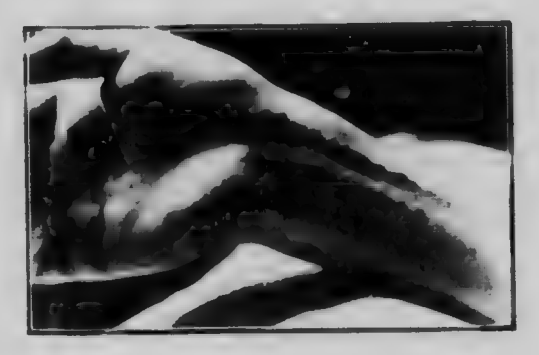

Fig. 24.

RHEUMATIC NODULES ON TENDONS OF THE WRIST.

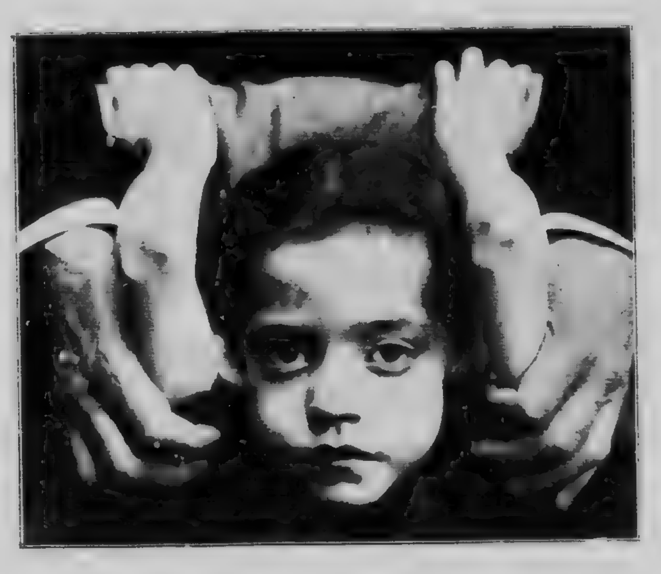

Fig. 25.

RHEUMATIC NODULES ON ELBOWS.

carditis. A great many cases of heart disease come before you in later life in which you can obtain no definite history of rheumatism in childhood, and many of these, no doubt, are cases in which there have been these fugitive muscular pains, which have been so trivial that no notice has been taken of them until the heart has become seriously damaged. These pains are particularly apt to affect certain parts of the body. I think you will find that the tendons of the hamstring muscles are more commonly involved than any other part, and that may lead you into errors of diagnosis. For instance, you may think that you have to do with a case of spastic paraplegia because the child walks on his toes, with the knees slightly bent, in order to spare the tendons; or you may suspect some bone trouble or some affection of the knee-joint.

The appearance of **nodules,** the third manifestation I have mentioned, is almost peculiar to early life. I think I have only twice seen rheumatic nodules in grown-up people, whereas in children they are of extreme frequency. Probably many of you are familiar with the appearance of these nodules; they are little things, varying in size from a pin's head to the size of a pea or bean. They tend to occur under the skin where the bones come near the surface. Thus you find them over the olecranon and the patella, or on the spines of the vertebræ, down the centre of the back. You may discover them also forming a festoon around the edge of the scapula, or dotted along the occipital suture at the back of the skull. You may also sometimes find them over the tendons. Presently I shall show you by means of lantern slides some nodules in one or two of these situations. But I want to impress upon you that they require to be carefully looked for. They are often more easily felt than seen, and they are apt to escape your observation. They are characterized by being perfectly painless, which may surprise you, and by being extremely mobile. When you cut into them you find they consist of little nodules of fibrous tissue, the fibres being arranged concentrically round a core of fibrin. Those nodules are not in themselves of any importance whatever; it does not matter if there are little fibrous lumps under the skin here and there; but, like those rheumatic pains which we have been considering, they are of the greatest importance as danger-signals, because they tend to go along with the heart complications, with endocarditis and pericarditis, and if you find rheumatic nodules coming out you may be fairly certain that there is progressive heart mischief going on. Dr. Cheadle said that when these nodules become really large they are practically equivalent to a sentence of death, because their significance as regards the state of the heart is so serious. You may compare them, if you like, to the vegetations which form on the edge of the valve in the heart. Just as in endocarditis of the mitral valve vegetations form along its edge, so in the case of nodules there are analogous fibrous deposits under the skin; but in the one case you speak of a vegetation and in the other case you speak of a nodule. Pathologically they are very much the same thing in the two cases. In the case of the nodules you may say they are vegetations which you can see and feel, while you may call the vegetations on the valves of the heart nodules if you like.

The fourth group of manifestations are those which occur in the **heart.** These, I need hardly say, are the most important of all. It is because their results are incapable of being entirely compensated for—because a valve once attacked is damaged irretrievably—that these forms of rheumatic manifestation are so serious. The commonest of them is endocarditis associated with pericarditis, and you may take it that in the majority of children who suffer from rheumatic heart affection these go together. You may not always be able to recognize pericarditis, but there is usually some degree of it present if there is endocarditis; and probably the converse is also true. You know that of recent years great attention has been paid in the pathology of the heart to the myocardium, and to the study of the changes which it undergoes in disease. Now, a study of the myocardium in acute rheumatism shows that it also is apt to be the seat of damage. You find small-cell infiltration between the muscle-fibres; you find degeneration of the latter, and you find later a development of fibrous patches.

It is because of these three things tending to occur more or less together—endocarditis, pericarditis, and myocarditis—that some people prefer to speak of the heart affection of rheumatism as simply '**carditis,**' meaning an inflammation of the heart in all its parts; and that is what you have to deal with in the majority of cases. But all the valves of the heart are not equally subject to attack in rheumatism. You may take it that in most cases the lesion which results from rheumatic heart affection in early life is mitral stenosis; and in the majority of cases also that is associated with more or less adhesion of the pericardium. Certainly in fatal cases these are the two changes which you find almost constantly: vegetations along the edge of the mitral valve, and more or less adhesion and fluid in the pericardium. Many children will escape those rheumatic manifestations as far as they affect the heart; and it will depend to some extent upon you how many of your patients so escape, because the better you are at detecting rheumatism the more easily you recognize its trivial manifestations, such as growing pains, and the more seriously and energetically you treat them the less will be the tendency for the heart to be affected; and by so doing you will prevent a great deal of subsequent cardiac disease in later life.

In the majority of cases you will find that the first attack of rheumatism does not permanently damage the heart. It is only after the second or third attack that the heart becomes the seat of lasting disease. It is true that in the first attack you may hear a whiffing systolic murmur at the apex, and you may therefore conclude that you have to do with damage of the mitral valve, which will go on to mitral stenosis or incompetence; and it is perfectly true that in such a case you *may* have to do with permanent damage of the valve. Yet there is no doubt that in many cases the murmur disappears entirely later on, and the heart becomes, so far as you can tell by examination, perfectly healthy once more. But if there is a repetition of the attack for a second or a third time the damage to the heart is almost certain to become permanent.

I now pass on to consider **erythemata** as a sign of rheumatism. An erythema is, as you know, a red eruption which appears like a blush over part of the skin, accompanied by more or less local heat, and which fades away again in a short time ; and there are some particular forms of erythema which are almost peculiar to rheumatism. One of them is erythema nodosum ; I think everyone will admit that it is related to rheumatism. It consists of an eruption of spots, slightly elevated, and usually more or less round or oval, which tend to appear particularly on the lower extremities, over the shins or calves, and are slightly tender. The skin over them is glazed, and they are often accompanied by a local inflammation of the ankle-joint. Sometimes there is a hæmorrhagic tendency in these cases, so that you get not merely erythematous patches, but actual hæmorrhages into the skin, and to that form the term *peliosis rheumatica* is applied. Then you may get large extensive areas of erythema over the trunk, a condition which is often spoken of as *erythema marginatum*. You will find that it tends to occur in circular patches with crescentic outlines. It may involve almost the whole body at one time, and may be, and not uncommonly is, accompanied by rheumatic manifestations in the heart.

Of **chorea** I do not intend to speak in detail. It is a disease which you have plenty of opportunity of seeing in the wards, and it is hardly necessary for me to take up your time with a description of it. I shall only say that as regards its relation to rheumatism most people believe that in about 75 per cent. of the cases of chorea there is an undoubted rheumatic association. Some people say it is invariably rheumatic, because you must remember that chorea is only one event in the series. It may be the first event, and if you follow the course of that child out you may easily find that later on he develops so-called growing pains, or joint inflammation, or endocarditis. **Pleurisy** also I do not require particularly to describe, because we shall come to it in another lecture ; but it is not a common manifestation of rheumatism, and when it is rheumatic in its origin it is probably usually of the nature of a dry pleurisy, or one without much effusion. Seeing that the pleura is of the nature of a large fibrous membrane, one would expert it to be more commonly attacked by the rheumatic virus than it is. But it is not nearly so susceptible as many other fibrous membranes. The reason for that I do not know.

Tonsillitis in its relation to rheumatism is a subject about which it is very difficult to speak definitely, because I do not think one can say that there is any given type of inflammation of the tonsil of which you can assert, simply by looking at it, that it is rheumatic in origin. On the other hand, no doubt, if you go into the history of children who have suffered from other manifestations of rheumatism you will often get a report of repeated sore throats. It may be that these are simply children with unhealthy throats, that there is follicular tonsillitis, and that the rheumatic poison gets access to a throat previously diseased. In purely rheumatic cases I think you will find that the inflammation is not confined to the tonsils, but tends to spread to the soft palate, and that it particularly tends to involve the tendons of the muscles, the tensor palati, and the levator palati, and to produce pain in upward and downward movement. At all events, one expects, here as elsewhere, to get the fibrous tissues involved rather than adenoid tissue, which is, as you know, the main constituent of the tonsil.

No matter which one of those manifestations has appeared, after it has lasted for even a comparatively short time you will find that the child gets into a cachectic state—what one may call the **rheumatic cachexia.** It is recognized largely by anæmia. The rheumatic poison tends more even than most toxins to destroy the red blood corpuscles ; and if you trouble to examine the blood of a child during an attack of endocarditis or pericarditis you will find a great loss of blood corpuscles in a short time , in a few days their number may drop by a million. One has sometimes been able to recognize the development of complications in an attack of rheumatism by the sudden pallor which the **anæmia** produces. Constantly, when going round the wards, if you have a child who is suffering from a slight rheumatic affection with fever, you will be able to tell if pericarditis has developed from the sudden increase in the pallor of the face. The fresh outbreak of the rheumatic poison seems to produce a fresh destruction of blood.

Lastly, we shall consider what the **treatment of rheumatism** should be. I need hardly say you should do all you can to prevent the disease developing in those children who are predisposed to it by heredity and by their diathesis. I say that because, although rheumatism is certainly due to a micro-organism, there can be no doubt that such agents as chill have something to do with its production, probably acting by diminishing the resistance of the tissues for the time being. See, therefore, that these children are properly protected from cold, that they are suitably clothed, and that in particular they are not allowed to get wet feet and sit in school in damp boots, which is, I believe, a very common cause of rheumatism in childhood. Be careful of the rheumatic child also in the matter of bathing. Over and over again one has known rheumatism develop after sea-bathing, and after the injudicious use of cold baths. Cold baths are very excellent things, but you must not make a fetish of them ; and many cases of rheumatism in early life are set up by the tendency to 'harden' the child by making him take a cold bath in the morning. The next point in the prevention of rheumatism is that you must take all the minor manifestations of the disease seriously. You have to insist upon the child going to bed at the first sign of the disease, no matter how slight and trivial it may seem to be. You may be inclined to think that a little aching in one joint or a little rheumatic stiffness about the muscles of the neck or the back is not worth paying serious attention to, but if you know the child to be predisposed to rheumatism you must insist upon his being kept in bed. If that were done regularly I am certain that a great deal of serious cardiac disease in later life would be prevented.

In regard to the treatment of rheumatism when it is actually developed, your chief care must be to protect the heart. The rheumatic pains are inconvenient and uncomfortable, and so is pyrexia, but they are not in themselves dangerous ; whereas the least degree of endocarditis or pericarditis involves a serious risk. Further, thanks to salicylates, you have the joint affections under your control. It is unfortunate that salicylates seem hardly to touch the cardiac manifestations; in fact, some people think their introduction has increased the amount of cardiac disease rather than diminished it by enabling the subjects of rheumatism to get up and go about sooner than they would have been allowed to do in the old days. When the pains are over, you are apt to think that the disease is over, and to overlook the fact that there is a slight endocarditis going on, which, if it becomes established, ends in permanent damage to the valves. So that what one has to preach about the treatment of rheumatism is the necessity of *rest.* One would sometimes like to put these cases in double long splints and keep them on their backs for six months! I am sorry to say that of necessity in such a hospital as this one has to set a bad example in this matter, for we cannot keep these cases in long enough; but you have to impress upon the parents that they absolutely must allow prolonged rest so as to prevent the heart becoming permanently damaged later on. If you insist upon prolonged and absolute rest, and at the same time use salicylates and iodides for the local affection, applying blisters over the heart when there are signs of pericarditis, you will do a great deal to prevent your patients becoming permanently crippled, and you will consequently diminish the amount of cardiac disease which one sees in grown-up persons.

LECTURE XV

THE RESPIRATORY DISEASES OF CHILDREN

GENTLEMEN,—In this lecture we come to the consideration of the respiratory diseases of childhood. Of course, I need hardly remind you that this is a large subject, and so in dealing with it it will be well for us to concentrate our attention chiefly upon those diseases of the respiratory system which are commonest in early life, or peculiar to it, and, for the rest, to glance simply at those points in which the respiratory diseases of children differ from the same affections as exhibited in grown-up persons. And in taking this course I must run the risk of sometimes appearing to be rather fragmentary.

I must remind you, in the first place, of certain **peculiarities of the respiratory organs** in children to which I directed your attention in the first lecture. You remember the peculiar shape of the infant's chest, how it is circular instead of being oval, as it is in the adult, one result of which is that the child's lungs can only expand easily in a vertical direction, and not nearly so much from side to side as do the lungs of adults. It is, perhaps, in consequence of this that collapse of the lower lobes of the lungs in children is apt to be rather common. I would remind you also of certain changes in shape which the chest may undergo in consequence of disease. These changes are rendered possible by the fact that the wall of the child's chest is soft and its cartilages yielding, the result of which is that they get driven in easily under ordinary atmospheric pressure when there are abnormal conditions present in the lungs. I would remind you of the rickety-shaped chest. You will remember that a falling in takes place at each side along the line of

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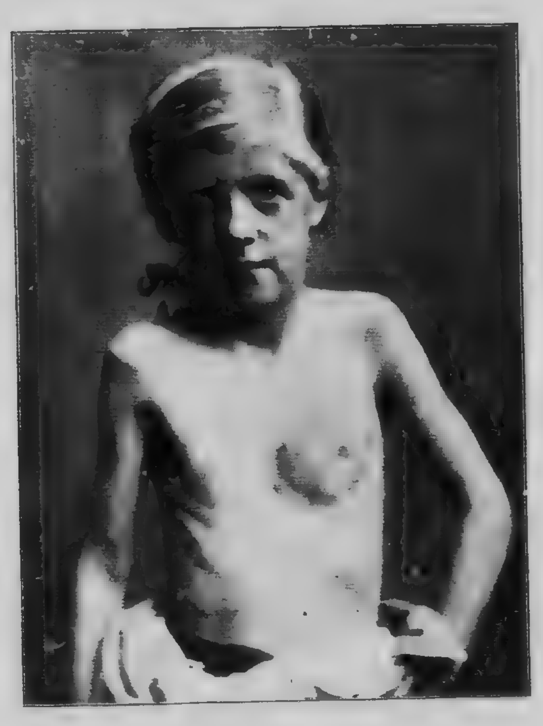

FIG. 26.

ADENOID CHEST, SHOWING DEPRESSION OF XIPHI-STERNUM.

attachment of the ribs to the cartilages ; and I would again direct your attention to the pigeon breast, which you must distinguish from the rickety chest, and in which the chest wall comes to a peak or keel, as it were, in front. That condition is commonest in diseases in which there has been a great strain thrown upon the chest for a long time, and in which there has been some consolidation of the lower lobes of the lungs, particularly, perhaps, after whooping-cough. There is a third type of chest which you should be familiar with, and that is the adenoid chest, which occurs in older children. Its chief peculiarity is a sinking in of the xiphisternum and lower costal cartilages. Later I shall show you illustrations of it with the aid of the lantern. Then there are certain peculiarities of the physiology of respiration in children. There is the great ease with which respiration in children becomes irregular, and in which the rhythm becomes altered on the advent of any acute disease, with the appearance of the inverted rhythm which I described to you before. I would remind you also that the breath-sounds are extremely harsh, that puerile breathing is the normal, and that on account of the great conductivity of the child's chest you may get accompaniments produced on the one side heard on the other side, and in consequence of which the breath-sounds are apt to penetrate to the surface of the chest, even although there may be a layer of fluid of some thickness between the lung and the end of the stethoscope. All these points should be borne in mind when you are studying respiratory disease in young children.

DISEASES OF THE LARYNX.

We will now look at the respiratory diseases in detail, and I shall begin with the diseases of the larynx, because in young infants they are very important and often very fatal. You will meet in practice with a group of cases, usually in children between the ages of one and five, in which the most striking symptom is **stridulous or 'croupy' breathing.** These cases are much commoner than you might suppose from your observation here, for but few of them find their way to hospitals. You will find, too, that most of these cases are spoken of by parents as 'croup.' That, gentlemen, is a very bad term, and one which I would advise you to avoid the use of as far as you can. Few terms in medical nomenclature have been the cause of greater confusion in teaching and reading than this word 'croup,' for it has been used without discrimination by different writers to designate totally different diseases. What I wish you to grasp quite clearly is that croupy breathing means simply laryngeal obstruction, and that such obstruction may be due to one of two distinct pathological processes :

1. To mere catarrh, with or without transient exacerbations of the obstruction from muscular spasm.

2. To membranous exudation in the larynx, which is practically always diphtheritic.

We shall devote our attention chiefly to cases of the first group, for you have opportunity of becoming familiar with diphtheria elsewhere.

One can distinguish two varieties in the **catarrhal cases,** which, however, are by no means sharply marked off from each other. These are (1) mild cases of laryngeal catarrh, in which there occur temporary exacerbations of the obstruction from muscular spasm ; (2) more severe cases, in which the swelling of the laryngeal mucous membrane is sufficient to oppose a more or less permanent obstacle to the entrance of air.

It will be convenient to have separate names for these groups, and so, following Goodheart, we may speak of the former as cases of *catarrhal laryngeal spasm*, and reserve for the more severe cases the term *catarrhal laryngitis*.

The history of a case of **catarrhal laryngeal spasm** is usually this : A child of about three years old has a slight cold, and, perhaps, a rather metallic cough. He goes to bed, however, as usual, without much being thought of it. He falls asleep, but an hour or two later wakes up in an attack of dyspnœa, accompanied by crowing inspiration, and in a state of considerable fright. These symptoms last for from a half to three hours, when they subside almost as quickly as they began, and the child falls asleep again. Next day there is probably still some metallic cough, and the croupy attacks are apt to recur for two or three nights. Such a case is probably never fatal. **Catarrhal laryngitis** also begins with a cold, but the croupy breathing and dyspnœa steadily increase, and there are no intermissions as in the milder variety of the disease. At the same time there may be a considerable degree of fever. Such a case will probably last for a week or longer, and you must also remember that laryngeal catarrh may occur at the outset of other inflammatory diseases, such as measles, or, as in a case we had in the ward recently, pneumonia ; and you must take care not to overlook the signs of these if they are present. You should always look upon laryngitis in children seriously. It is by no means uncommonly fatal, and I have had the misfortune to see two cases die on the table whilst tracheotomy was being performed.

We come now to the important matter of **diagnosis.** Catarrhal laryngeal spasm is usually easy to recognize. In most of your cases there will be a history of previous attacks, and the sudden onset in the middle of the night is very characteristic. Catarrhal laryngitis in its severe forms is much more difficult to be sure of. Such cases will often cause you great anxiety at the outset, for you may have great difficulty in excluding **laryngeal diphtheria.** I believe, indeed, that the diagnosis between the two may sometimes be impossible, for the signs of obstruction are the same whether it be due to mere swelling of the mucous membrane or to membranous exudation, and there is nothing definite in the general symptoms to guide you. The only conclusive test would be a laryngoscopic examination, which it is, of course, almost impossible to perform. The chief points to be attended to, however, are these :

1. The more rapidly the symptoms set in, the more likely it is that you are dealing with simple and not membranous inflammation. As Trousseau has said, 'simple laryngitis makes a great fracas, but diphtheria insidiously instals itself.'

2. The voice is more apt to be lost in diphtheria than in laryngitis, and the cough to be less, for the membrane protects the mucous surface from irritation. Diphtheria, *in* short, is the more *silent* disease of the two.

3. The constitutional disturbance, but not the temperature, is usually greater in diphtheria, and the child looks more *ill ;* but this, of course, is an indication which it requires some experience to appreciate.

Enlargement of the glands in the neck is of doubtful value, for although that occurs in diphtheria, yet children who are most predisposed to laryngitis from an unhealthy condition of the throat are apt to have it too. This practical rule you will do well to lay to heart : *if in doubt regard the case as one of diphtheria, and treat it accordingly.*

Let us pass now to **treatment.** In dealing with a case of catarrhal laryngeal spasm you have to consider (1) what you should do when summoned during an attack ; and (2) what steps you are to take in order to prevent recurrence.

When confronted with a case of catarrhal laryngeal spasm you will remember that, no matter how alarming the symptoms may appear, such cases are never fatal, and so you will keep calm and endeavour to allay the natural anxiety of the friends. Your next step should be to administer an emetic. You will find powdered ipecacuanha the best for your purpose. Give a 10-grain dose and repeat it in a quarter of an hour if necessary. Ipecacuanha wine is often recommended, but it is a much less trustworthy preparation. I have given as much as an ounce of it in teaspoonful doses every quarter of an hour without producing emesis, but if you get a fairly fresh preparation of the powdered root you will rarely fail with it. The next thing to do is to apply hot fomentations over the larynx and to arrange some method of inhalation. The best plan is to rig up a tent over the child's cot and to let the steam from a bronchitis kettle play into it. You may use a little creosote with the kettle if you like, but the great thing is to secure the local internal application of moist heat. These measures will usually be speedily successful, but if you are dealing with a severe case it may be justifiable to administer a whiff of chloroform to allay the spasm. In order to prevent the recurrence of an attack the following night you should continue the administration of small doses of ipecacuanha throughout the next day, and at bedtime give a dose of antipyrin, 1 grain for every year of the child's age.

As most of these children are predisposed to their attacks by having enlarged tonsils or adenoids, it is well to attend to the condition of the throat in order to prevent the reappearance of the disease. Cold also should be guarded against, for it is a potent exciting cause. In Scotland attacks of 'croup' (as it was called) used to occur with special frequency on Saturday nights, the probable explanation being that nursery floors were usually washed on that afternoon in preparation for Sunday, and the attacks were brought on by children being put to bed in a room which was rather damp.

In the treatment of **acute laryngitis** I would remind you again that if you have the least suspicion that the case may be one of diphtheria it is your duty to give an injection of antitoxin, and to take all the precautions usual in dealing with the more serious disease. The rest of your treatment must proceed much on the lines already laid down for catarrhal laryngeal spasm. Inasmuch, however, as the obstruction here is organic and not spasmodic, you will expect less benefit from emetics. Should the case be a very 'florid' one—*i.e.*, the temperature high and the distress great—the old so-called 'antiphlogistic' treatment often gives very gratifying results. It consists in blood-letting either from the jugular vein or by the application of a couple of leeches above the sternum. A full dose of calomel should also be given and antimonial wine administered in 5-minim doses every two hours. If the dyspnœa increases you must be prepared to perform intubation or tracheotomy, and it is well to remember that it is safer to operate too early than too late. Of the relative merits of the two procedures it is scarcely my province to speak, but I would only say that I believe nearly all the advantages lie with intubation, and I should greatly like to see it more widely used in this hospital.

Other laryngeal diseases in children about which you may make mistakes are **congenital laryngeal stridor** and **laryngismus stridulus.** These are totally different diseases, and I shall have occasion to describe them when I come to speak of the neuroses of children, so I only mention them here for the sake of completeness. There is another condition which you should bear in mind when a child has dyspnœa of laryngeal origin, and that is the possibility that you are dealing with papilloma of the larynx. That occurs not uncommonly in children; more commonly, I think, than in grown-up people, and oftener in boys than in girls. The child is usually about three or four years of age, and the symptoms simulate those of chronic laryngitis, for which papilloma is apt to be mistaken. So if you have a child brought to you with a husky voice and occasional slight dyspnœa, and a history that the symptoms have lasted for weeks or months, you should think of laryngeal papilloma. I think you can make an exact and certain diagnosis only by the use of the laryngoscope, and that is a matter of such difficulty that I advise you to send those cases to specialists; in any case you will require a specialist to operate upon them, because the only successful treatment is removal of the growth with forceps through the mouth, which is a very delicate and difficult procedure.

BRONCHITIS.

I now pass to the consideration of a disease which is extremely common in children—namely, *bronchitis*. Bronchitis is certainly commoner in childhood than at any other period of life, and the reason for that may be the tendency of children to get chilled owing to their large body surface; contributing causes certainly are rickets and the occurrence of teething. You constantly find bronchitis as one of these catarrhal manifestations of rickets of which I have already spoken, and you will find children who get an attack of bronchitis every time they cut a tooth. Why that should be I do not know. Some people say it is produced in some mysterious reflex fashion, but I think it is more likely that the teething simply lowers the child's resistance, like any other agency, and so renders it specially liable to suffer from chill.* Now, 'bronchitis' in itself is never a sufficient diagnosis. That is true not only of children, but of grown-up people. But it is particularly true of children, because they are apt to suffer more than grown-up people from capillary bronchitis—that is to say, inflammation of the small tubes, which may be a very serious matter. And so, having established the fact that the disease exists, you have to ask yourselves two questions: first, Which set of tubes is involved—the large tubes or the small ones? and, secondly, What stage has the bronchitis reached—is it early or is it late? With regard to the first of these questions, you will settle whether you are dealing with bronchitis of the large or of the small tubes by the character of the accompaniments. If the **large tubes** are involved you will hear low-pitched accompaniments—*i.e.*, sonorous rhonchi—if it is in the dry stage, or more or less coarse bubbling sounds in the moist stage; whereas if the **small tubes** are involved the accompaniments will be high-pitched rhonchi if the bronchitis is dry, or fine, crackling sounds if it is moist. Again, in a case in which the fine tubes are involved the accompaniments are far more numerous than in those cases where it is the large tubes which are chiefly affected. The obvious reason for this is that there are far more small tubes than big tubes in the lung, so inflammation of the small tubes will result in your being able to hear many accompaniments; and if you hear rhonchi coming from every quarter you can be certain you are dealing with inflammation of the small tubes. The other question is, What **stage** has the disease arrived at in your case? That is settled by the character of the accompaniments—whether they be dry or whether they be moist. In the early stage of the disease you have dry sounds, and in the later stage the sounds become both dry and moist, whereas in the latest stage the moist sounds predominate. The question of whether the large or the small tubes are involved in the inflammation is one of great importance in the prognosis, and that is chiefly why you wish to determine that point. You take a more serious view if the small tubes are chiefly involved, because the great thing you have to fear in a case of bronchitis in a child is that the condition may pass on to broncho-pneumonia—that is to say, to inflammation of the lung substance—instead of being merely an inflammation of the lung tubes. The remote danger in bronchitis, you will remember, is that it leaves behind an unhealthy condition of the bronchial glands, which renders them liable to become the seat of tuberculosis. With regard to bronchitis, I would further say that you should always take a more serious view in a case in which the chest wall is very soft and yielding. Practically it amounts to this, that bronchitis in a rickety child is a far more dangerous affection than a similar degree of bronchitis in a child who is otherwise in good health. The rickety chest is so soft that it readily allows collapse of the lung, and the extension of the process to the collapsed lung, with the production of pneumonia.

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* In some cases it may be due to the clothes over the chest getting wet from the 'dribbling' which accompanies teething.

The **treatment of bronchitis** must depend upon the stage at which the disease has arrived. If you are dealing with an early case where there is fever, more or less dyspnœa, rapid breathing, and high-pitched dry sounds, then you ought to put the child in a tent, and let it have creosote inhalations. That is probably far more valuable than the administration of any amount of expectorants by the mouth. Here at this hospital, in the out-patient room, you get a wrong idea of how to treat these cases ; you cannot get out-patients to use bronchitis-kettles, and so one has to fall back on the second-best treatment—namely, the internal administration of expectorants. But when you have complete control of the case the best plan is to put the child in a tent, and fit up a bronchitis-kettle, and use creosote as an inhalation along with the steam. It is well also that those cases should have either a cotton-wool jacket or a poultice over the back. It is a mistake to put poultices over the front of the chest, because they are too heavy ; they increase the labour of breathing, and the child ought not to have anything to restrict the breathing. But a poultice over the back is often very valuable. You may use for the poultice linseed-meal and mustard. There is no harm in giving, in the early stage, small doses of ipecacuanha by the mouth along with alkalies, such as citrate of potash or acetate of ammonia. And such children should be encouraged to drink large quantities of hot fluids, because there is nothing better for promoting the secretion from the bronchial tubes than the administration of hot fluids of all sorts. That is the meaning of the old domestic teas which used to be brewed. They act, not in virtue of the material in them, such as camomile, but simply in virtue of the fact that you are giving large quantities of hot liquid. In the later stage, when you have got to deal with much liquid mucus in the tubes, you will find it an advantage to administer an emetic, particularly where the child has difficulty in getting rid of the mucus, and where there is much choking and a furred tongue. There are one or two conditions which contra-indicate the giving of emetics. One of these is the presence of much cyanosis. The reason is that in that condition the nervous system is, as it were, partially narcotized ; it does not respond to the irritation of an emetic, and you are apt to have the ipecacuanha or other emetic retained, and great depression result without emesis. Great prostration is the other condition which contra-indicates the giving of an emetic in the second stage. In the last stage—that is to say, when the patient is convalescent—you should administer such things as iron and cod-liver oil. and apply stimulating liniments to the chest until all accompaniments have disappeared. You must make convalescence complete because of the great danger of tuberculosis starting in the bronchial glands, which have become chronically inflamed.

ASTHMA.

Along with bronchitis one may conveniently consider asthma. Asthma is not uncommon in children—even in infancy—but boys suffer more often than girls. It tends specially to occur in those of gouty or neurotic stock, and a history of attacks in the near relatives, or of other paroxysmal neuroses, such as migraine, is not uncommon.

One can recognize, I think, four types of the disease : (1) Ordinary typical asthma, such as is met with in the adult ; (2) attacks which more resemble hay-fever ; (3) 'bronchial-asthma '—*i.e.*, recurring bronchitis with a spasmodic element superadded ; (4) the bronchitic type, in which the attack resembles one of very acute capillary bronchitis, but with an unusual amount of wheezing and dyspnœa, and which begins and ends abruptly.

In all cases when the disease has lasted for some time an element of emphysema is apt to develop, which adds to the gravity of the condition, and if the attacks be frequent and severe the child is prone to be stunted in growth and development.

You will not have much difficulty in the diagnosis of asthma except in cases of the bronchitic type, in which there is a danger of the asthmatic element being overlooked, especially when the patient is a young infant. The abrupt subsidence of the attack is, however, characteristic. This is the only variety of the disease, also, in which there is any real danger from the attack itself, for in delicate infants it may prove fatal; in the other types the chief prognostic interest is as to whether the attacks are likely to persist into adult life. As to this, one should speak with caution. My own experience—and I have followed some of my cases for more than ten years—is that *complete* disappearance of the attacks is exceptional, though they often become much less frequent and severe. If emphysema has developed, the outlook is always more serious, although the slighter degrees of emphysema may be recovered from if the attacks disappear.

In treatment you have to consider (1) what to do in an attack; (2) how to ward off attacks; (3) how to treat the tendency to attacks.

1. **In an attack** the inhalation of the fumes of one of the well-known anti-asthmatic powders is helpful, the fumes being introduced under an improvised tent. The only disadvantage of this method of treatment is that it favours the development of bronchitis, as the fumes are always rather irritating.

If the stomach be overloaded, an emetic will often cut short the attacks, and is always worth trying.

If the distress be very severe, you may be compelled to inject morphia ($\frac{1}{16}$ to $\frac{1}{4}$ grain, according to age). Adrenalin injections (3 to 5 minims of a 1 in 5,000 solution) are also very successful in some cases.

2. The best way to ward off an attack is to give a dose of antipyrin (5 grains or more), or a sedative dose of chloral or bromide at bedtime, and to see that the child goes to bed on an empty stomach.

3. The treatment of the underlying tendency is the most important and difficult part of your task in the management of asthma cases.

In the first place you should look for any source of **reflex irritation,** and first of all in the naso-pharynx. Nasal obstruction, adenoids, or enlarged tonsils, should always be dealt wit The immediate effect is often most striking, but, unfortunately, the paroxysms tend to recur again later on, although often in a milder form than before, and in some instances they disappear permanently after the operation. The bowels should be regulated in order to prevent reflex irritation from the intestine, and it is considered by many advisable to perform circumcision if there be any phimosis.

The **diet** may need to be modified. Starchy foods should be cut down, and care taken that the stomach is not overloaded. You should remember in this connection that asthmatic children often exhibit curious idiosyncrasies to certain foods—eggs for example—and it may be necessary to eliminate these from the diet.

Hygienic treatment consists in avoiding cold and damp as far as possible. The feet and legs should be kept warm and dry, and the chest and neck protected. Sponging of the chest with cold water after the daily warm bath is believed to be helpful in 'hardening' the child against bronchitis. A thorough course of respiratory exercises is often very useful.

Climatic treatment is of the very first importance, but your great difficulty will be to know where to send the child and what sort of climate to advise. There is, I am sorry to say, no invariable rule to guide you, for every asthmatic is a law unto himself. In one of my cases, for instance, the child was quite well when in London, but had frequent attacks when at home at Lowestoft; another boy remains quite well when at school at Eastbourne, but invariably relapses when he returns home to Blackheath; a third, a girl, is a chronic asthmatic when in Croydon, but when she goes away from it is immune, and this peculiarity has persisted now for ten years and more. In view of such inexplicable peculiarities one can only advise the method of experiment—trying the child in various places, and if one is found where the attacks disappear, keeping him there. This, however, is not always feasible from the family point of view. When there is much coexisting bronchitis you need never hesitate to advise a warm, dry climate as the best, if circumstances permit.

The most valuable drugs in the treatment of the asthmatic tendency are iodide of potassium (in small doses), ethereal tincture of lobelia, stramonium, and arsenic. These may be given in combination, but the whole secret of using them is *to persist in their administration for months,* just as one uses the bromides in the treatment of epilepsy. It is in such use that the best hope of overcoming the tendency to the attacks resides, and you must urge parents to persist in the treatment with patience.

LECTURE XVI

RESPIRATORY DISEASES—*Continued*

PRIMARY OR CROUPOUS PNUEMONIA IN CHILDHOOD

GENTLEMEN,—Attempts to classify cases of pneumonia in children have given rise to a considerable amount of confusion, and, indeed, one may admit frankly that it is often a very difficult matter at the bedside to refer any given case to a particular division of the disease. Hence for practical purposes I think it is best to keep one's divisions as broad as possible, and I therefore propose to divide cases of pneumonia into those which are primary on the one hand, and those which are secondary on the other. By **primary pneumonia** I mean a case in which the inflammation starts in the lung substance ; by **secondary pneumonia** I mean that form of pneumonia in which the inflammation of the lung-substance is consecutive to disease of the air-passages, usually to bronchitis. Hence the term 'broncho-pneumonia' usually applied to it. Further, you may contrast primary and secondary pneumonia in this way: that primary pneumonia may be either lobar or lobular, although it is more commonly the former ; whereas secondary pneumonia is usually, perhaps one may say always, lobular. In the second place primary pneumonias are practically always due to the pneumococcus, whereas secondary pneumonias may be due to many organisms, perhaps most commonly to a streptococcus. When you come to ask yourselves which form of pneumonia is the most frequent, it is very difficult to answer, because it depends very much on the type or class of case which you see. In this hospital, where we do not treat cases of the acute infectious fevers, there can be no doubt that primary pneumonia is far commonest; whereas if you were to go to one of the Metropolitan Asylums Board Hospitals, where there are cases of measles, scarlet fever, diphtheria, etc., you would probably find a large number of cases of secondary pneumonia, because secondary pneumonia is a common complication of many of the acute infective fevers. But here in this hospital, and I would say further, in ordinary general practice, you will meet with very many cases of primary pneumonia in children of all ages. I say it is met with in children of all ages, but it is most frequent, as statistics show, between one and two years of age. That is the period of maximum incidence of primary pneumonia in childhood, for about that age children seem to be peculiarly susceptible to the attack of the pneumococcus. I have recently gone through the statistics of seventy cases of primary pneumonia which I have had under my care in children below seven years of age, and I found that half of these were below two years of age.

MODE OF ONSET.

As regards the onset and symptoms of primary pneumonia—and it is only with primary pneumonia I shall deal this afternoon—it may begin sometimes in a very deceptive way. It is not uncommon, for instance, for it to begin with **vomiting.** A child may be taken with severe vomiting, along with rise of temperature, and for the first few hours these may be the only prominent symptoms. A mode of onset like that is apt to deceive you. It makes you think perhaps of brain trouble, such as meningitis. Or it makes you think of some of the acute fevers, particularly scarlet fever, which you know often begins with vomiting. Another deceptive mode of onset of pneumonia is with **drowsiness.** I have known cases in which, even for a day or two preceding the actual onset, there was great drowsiness. I remember, for example, the case of a little boy of whom it was said that a day or two before he was ill he was always falling asleep at school; apparently before the disease had made a proper beginning that symptom had arisen. As a rule, however, the disease sets in fairly abruptly, with a rise of temperature and a certain amount of shivering. A true rigor is not so common as it is in grown-up persons ; but a certain amount of shivering is not rare. And sometimes in sensitive nervous children these shivering fits take the form of a slight attack of convulsions. Very quickly you will notice an alteration in the character of the respiration, and that is one of the most important things for you to observe, because it is upon the altered character of the respiration that you will sometimes have to depend for making your diagnosis in the early stages. And the alteration is this : In the first place the respirations are greatly increased in frequency out of proportion to the increase of the pulse-rate. Secondly, the respiration takes on that character which I have often demonstrated to you in the out-patient department as 'inversion of the respiratory rhythm.' In the normal state of things the respiratory rhythm goes thus : inspiration, expiration, pause ; but in pneumonia, and indeed in all grave respiratory affections in young children, there tends to be an inversion of the rhythm, so that one gets expiration, inspiration, pause ; and you will remember that the expiration is attended often by a curious grunt, and during the grunting expiration the alæ nasi are blown out, which is again an inversion of what ordinarily happens in an adult with dyspnœa, in whom, if the alæ nasi are acting, they open out during *inspiration*. Upon this altered character of the breathing, as I have said, you may have to depend in the early stages for making your diagnosis, and therefore it is extremely important that you should be familiar with these respiratory changes. Quite early in the onset of the disease you will notice that the child is flushed, and not uncommonly you find a certain amount of herpes. But in a large number herpes is absent, so you must not depend upon it at all as a diagnostic sign ; it is too often absent to be of use in that respect.

PHYSICAL SIGNS.

The unfortunate thing about the physical signs of pneumonia in children is that they are often absent at the outset and for some time after the disease has fairly begun, and, indeed, you may find them only becoming marked when the crisis is at hand. I have even notes of cases in which they become evident only *after* the crisis. This is no doubt due to the inflammation starting deep in the lung and only by degrees reaching the surface.

The most important physical sign is an impaired note on percussion, and I am constantly pointing out in the out-patient department that if you wish to elicit this impaired note you must percuss properly and especially lightly. Too much stress cannot be laid on the word 'lightly.' Repeatedly one sees areas of pneumonia missed on physical examination because the observer percusses so forcibly that he elicits resonance underneath the patch of pneumonia altogether. You cannot, therefore, be too delicate in your percussion if you are to make out the signs in an early case. It is important to know where to percuss, and that will depend on the sites at which pneumonia is commonest. Pneumonia in children differs from pneumonia in the adult in this, that the apex is relatively much more often affected than it is in grown-up people. I have found that out of my cases, for instance, one-third of the total were apical cases, cases in which the upper lobe of the lung was involved. That is a far higher proportion of apical cases than in the adult. Apical pneumonia in the adult is the exception, but you cannot say that of pneumonia in the child. Further—and this is a curious and unexplained fact—of the apical cases two-thirds occurred in the right lung. I think you may say that the most common situations for pneumonia in the child are the left lower lobe and the right upper lobe, and therefore it is in these situations in a doubtful case you will percuss with the greatest care. In particular I wish to commend to you the importance of percussion below the right clavicle. I have known any number of cases of pneumonia overlooked because the observer had failed to percuss lightly below the clavicles. Apart from percussion, the other chief sign is an alteration in the breath-sounds, the development of what is known as tubular breathing—that high-pitched whiffing kind of breathing which occurs over consolidated lung. That is a sign which develops sooner or later in all cases, but it does not develop quite so soon as the impairment of note, and so is of less value than percussion. These are the only methods of physical examination which you need employ—delicate percussion and auscultation to determine the presence or absence of tubular breathing.

SYMPTOMS.

Compared with the adult, the symptoms are far less pronounced. You will often see—I saw one the other day—a child with full-blown pneumonia standing up and prancing about in its crib, apparently not much the worse for the fact that it has a temperature of 103° F. and consolidation of the lobe of one lung. You do not see that in the adult. A grown-up person with lobar pneumonia is very ill indeed, whereas a child with the disease may not appear to be seriously ill at all. The reason for this is that the toxæmic symptoms which are so very serious and prominent a feature in pneumonia in the grown-up person are comparatively absent in a case of pneumonia in childhood. For some reason, which we do not know, children do not seem to suffer from the toxins of pneumonia in the way that the grown-up person does. Delirium, for instance, which is one of the most prominent symptoms of toxæmia in the adult, is a very rare thing in the child, and, on the other hand, heart failure, one of the things which gives you the greatest anxiety in the pneumonia of a grown-up person, is something which in the child you need hardly fear at all. So that the two great groups of symptoms, the toxæmic or nerve symptoms and the symptoms which result from heart failure, are comparatively absent in children, and for that reason the symptoms, apart from the feverishness and the hurried respiration, may be very trivial indeed.

MODE OF TERMINATION.

As you know, pneumonia of the primary sort in children, whether it be lobar or lobular, terminates, as a rule, by crisis. You may put that in another way, and say that pneumococcal pneumonia in children tends to be a self-limited disease, as distinct from streptococcal and other forms of pneumonia which are not self-limited, but drag on for an indefinite time. In the majority of cases the crisis occurs on the seventh or eighth day. That has certainly been so in the majority of my cases. But in a considerable number of children the crisis occurs earlier, on the fifth or sixth day, and in a comparatively small number after the eighth, say on the ninth or tenth day. It is rather characteristic of pneumonia in children that the crisis is apt to be preceded by one or more sudden falls of temperature, which are spoken of as 'pseudo-crises.' You can distinguish a pseudo-crisis from a real one by the fact that after a true crisis the respiration-rate falls, but in a pseudo-crisis the respiration-rate is not affected. So the respiratory chart is really a better guide to the occurrence of the crisis than the temperature chart alone. Again, sometimes after the true crisis there is a temporary rebound of fever lasting a few hours, twelve hours perhaps, which may alarm you unnecessarily, and make you think that some complication is arising. These temporary rebounds after the crisis are not very uncommon in early life. The heat-regulating centres in children seem to lose their equilibrium more easily than in the grown-up person. If you set them into violent oscillation, it takes some time before they settle down again. And if the temperature drops to subnormal at the crisis, it is apt to jump up above the normal again, just as an india-rubber ball rebounds when thrown to the ground. But in a few hours it finally settles down to the normal. Sometimes, instead of a crisis occurring, the fever is prolonged, and it is about the causes of prolongation of fever in pneumonia that I now want to say a few words.

PROLONGED PYREXIA IN PNEUMONIA.

You may divide cases in which the temperature does not fall into, first, *protracted cases*—that is to say, cases of pneumonia in which, for some reason, the temperature runs on for ten, fourteen, or more days—I have even known it to go on for several weeks—without the disease spreading and without any complications arising, and for no particular reason which you can find. It seems in such cases that natural resolution is extremely slow, and I think they are apt to terminate in fibrosis of the affected part of the lung. Cases of fibroid lung affecting the upper lobe, which are sometimes met with in children, and which are not tubercular, probably have their origin in an attack of pneumonia of this sort, which has run a protracted course.

Secondly, the prolongation of the fever may be due to the *spreading of the pneumonia* or to its *relapsing*. That is to say, the disease spreads from one lobe to another, and the child may, as it were, run through two attacks of pneumonia consecutively. I have known practically every lobe of the two lungs to have been involved successively, each one giving rise to a further rise of temperature as it has become affected. Here is the chart of a case of protracted fever in pneumonia due to involvement first of the right upper and then of the right middle lobe, and it was a case in which, as you will see, the temperature ran on for considerably more than a fortnight. One speaks of these cases sometimes as 'wandering pneumonias'—the disease wanders from one lobe to another. The prognosis in these circumstances is not necessarily bad, but one is naturally always more anxious in such a case than in a straightforward pneumonia, in which one lobe only is involved. Thirdly, failure of the temperature to come down, or a tendency for it to rise after it has once come down, may be due to the development of *complications*. These complications are due to the starting of a focus of pneumococcal infection elsewhere, and in young children—and the younger the child the more this is true—there is rather a tendency for this generalization of the infection. Indeed, in quite young infants a condition of general *pneumococcal septicæmia*, as it is sometimes termed, is by no means uncommon. Of the possible complications, *empyema* is certainly the commonest. It is characteristic of empyema that after the crisis the temperature tends to remain down for a few days, and then gradually begins to rise again. You see it gradually rise until pus is found and evacuated, and then it comes down. The signs of empyema are the development of stony dulness, with very marked *resistance* on percussion : that is the chief point, marked resistance on percussion at the base of one lung. It may, however, be extremely difficult to tell an empyema with certainty from an unresolved pneumonia, or from one of those cases in which a thick patch of lymph forms over the pleura, and, indeed, there is only one way of diagnosing empyema with certainty, and that is by putting in an exploring needle; and if you find the temperature chart creeping up like that after the initial crisis and the dulness persisting, and still more, much resistance on percussion at the base, you should not delay exploring, because it is certain that if empyema is diagnosed early and evacuated, the prognosis is vastly better than if it is left till later. The cases of empyema which do badly are those in which the diagnosis has not been made until two or three weeks after pus has formed.

Another common complication is *middle-ear disease*—that is to say, an infection of the middle ear by the pneumococcus. I have known a case in which the temperature ran on after an attack of pneumonia for four weeks, and we could not discover what was the cause, until finally some pus was discharged from one ear, and after that the temperature came down and everything went well. Observe this about pneumococcal middle-ear disease, that it may not give rise to any local signs which would make you suspect its existence; the child may not complain of earache, and there may be nothing to put you on the scent as to the true nature of the trouble. So where there is a prolongation of the fever, and you can find no cause for it, do not omit to examine for suppuration in the middle ear. We omitted it in the case I spoke of, and that is why we did not find the cause of the rise of temperature. The third complication I have to speak of is a very serious one, namely, *pneumococcal meningitis*. The difficulty about meningitis is not so much to diagnose it when it is there, but it is that you are apt to think there is meningitis when it is not there; and you are apt to do that because several of the symptoms and signs of meningitis may occur in cases of pneumonia, especially, perhaps, in apex pneumonias, simply as a result of the pneumonia itself. They may also occur in middle-ear disease where there is no meningitis at all. I refer especially to such signs as vomiting and head-retraction, and even the development sometimes of Kernig's sign. All these may occur in cases in which there is no real meningitis, and you may often be in doubt whether you are dealing with an apex pneumonia which is simulating meningitis, or with pneumonia complicated by meningitis. The only way of determining this point in a doubtful case is by lumbar puncture, for that will enable you to draw off cerebrospinal fluid containing the pneumococcus, and so to establish the diagnosis.

Another complication is *pericarditis*, and a very serious one it is. But I do not propose to say anything about pericarditis to-day; I shall deal with it when I lecture on the acute cardiac affections of childhood generally. So I shall now pass to the next complication, and that is *pneumococcal peritonitis*. This is probably a commoner complication in pneumonia than is supposed. It is a complication which is rather apt to be overlooked, and it is only in comparatively recent times that its existence has been properly recognized. And yet pneumococcal peritonitis occurs by no means very infrequently in childhood, either as a complication or as a sequela of an attack of pneumonia. The usual symptoms by which it is ushered in are the development of abdominal pain, diarrhœa, and vomiting, and a rise of temperature if it has already fallen. Quite soon, too, retraction of the abdomen can be observed. These are the initial symptoms, and as effusion takes place you begin to find signs of fluid in the abdomen, and when that has occurred, many of the acuter symptoms subside, and the case becomes more like one of tuberculous peritonitis. The treatment of this complication is surgical.

The last complication I shall mention is *pneumococcal arthritis*. This is not a common complication, but it occurs every now and then, especially in quite young children, and it is a curious thing that it does not necessarily go on to suppuration. You may find that a joint, often one of the smaller joints, becomes red and swollen, and that it is hot and painful, and after a time the whole thing subsides, and no further harm results. Every now and then, however, suppuration takes place, and if this happens, the treatment adopted must be the same as in any other form of suppurative arthritis.

These, then, are the commonest complications of primary pneumonia in childhood. Sometimes several of them occur together, and it is not uncommon to have a child who has an attack of pneumonia developing middle-ear disease, and perhaps meningitis, both the result of the pneumococcus; or he may have pneumonia followed by empyema and an attack of pericarditis; or where there is generalized pneumococcal septicæmia, practically all these things may occur together. Such cases, I need hardly say, are almost inevitably fatal.

DIAGNOSIS.

As regards the diagnosis of primary pneumonia in children, I have told you that it is at the outset that your difficulties will arise. You are called to a child who has a sudden rise of temperature and some vomiting, and you are asked by the parents what is the cause of the attack. Naturally, you think first of the acute specific fevers, and the acute specific fevers which is most apt to simulate pneumonia is *scarlet fever*, because, as I told you before, scarlet fever often begins with vomiting and a sudden rise of temperature, just as pneumonia does; but, of course, in the latter you do not have a rash or sore throat, and further, whereas in scarlatina the rate of the pulse is specially raised, in pneumonia it is the respiration-rate which is specially affected, and attention to that point of difference will usually keep you right. Another thing which may simulate pneumonia is *acute abdominal disease*, especially appendicitis. This is now generally recognized, and I think all surgeons are alive to the possibility that a pneumonia involving the lower lobe of the right lung may simulate very closely an attack of appendicitis. Cases in which the surface of the pleura covering the diaphragm is specially involved seem to me to be those in which the simulation is most liable to occur. You will find a like simulation in cases of pericarditis, where the pericardium covering the diaphragm is involved; these also may simulate abdominal disease very closely. Hence, when you are dealing with a case of suspected pneumonia, with pain on the right side, and vomiting and a rise of temperature, you should always have in mind the possibility of appendicitis. Conversely, where there is doubt about an acute abdominal condition, remember pneumonia, and study carefully the character of the breathing, and examine for physical signs at the right base.

Lastly, pneumonia may specially simulate *meningitis*. I have already referred to that in speaking of meningitis as a complication of pneumonia, and I need not say anything further about the differential diagnosis.

PROGNOSIS.

As regards the prognosis in primary pneumonia in childhood, this may be said, that it is vastly better than it is in grown-up persons, and, indeed, I always regard an uncomplicated case of primary pneumonia in a child as a comparatively trivial matter, unless the patient be below one year of age. In early infancy even an uncomplicated case may be serious, but in children of two years of age or thereabouts uncomplicated pneumonia is almost never fatal. Of seventy uncomplicated cases below the age of seven which I have had under my care at the London Hospital, not one died. But when there are complications the case is different, for some of these are very serious indeed. The most dangerous complications are meningitis and pericarditis, but fortunately these are rare. In the absence of complications you may usually prophesy without fear that in about a week the disease will have terminated by crisis, and that the patient will get well.

TREATMENT.

In a case of uncomplicated croupous pneumonia there is very little you need do in the way of treatment, and, as a matter of fact, my own cases, except for nursing, which is very important, get really nothing which can be described as treatment at all, and I find that they do very well without it. If the child is restless and the temperature very high—that is to say, 104° F. or upwards—it is perhaps a good thing to reduce the fever a little by tepid sponging ; but there is no use in attempting to force the temperature down too far. Cases with high temperature do as well as, and, I think, better, than those in which it is low. You may also give a simple diaphoretic mixture to keep the skin and kidneys active.

Digitalis, which is so useful in the treatment of pneumonia in the adult, is hardly ever required in children, and I very rarely order alcohol. Nerve sedatives, also, are scarcely ever called for; but if want of sleep is a difficulty, a few grains of Bromural or of Dover's powder may be given with advantage.

As regards the general treatment, see that the room is kept well ventilated, that the child is not over-clothed or over-fed, and that he is allowed plenty of cold water to drink.

The treatment of complications I have already referred to.

LECTURE XVII

RESPIRATORY DISEASES—*Continued*

GENTLEMEN,—I wish to speak first to-day of **broncho-pneumonia.** This is a disease which you often see in the wards, one of very great fatality—so much so that about 17 per cent. of the total infantile mortality results from it. Broncho-pneumonia may be either primary or, more commonly, secondary, occurring in the course of another disease, such as measles or whooping-cough, and in such cases the broncho-pneumonia is very often the last straw, as it were ; it is that which induces a fatal ending. Now, your difficulty will always be, when you are in contact with a case of supposed broncho-pneumonia, to say when you are dealing with simple bronchitis and when the disease has passed on a stage further—namely, to broncho-pneumonia. I would like to emphasize the fact that **physical signs** will here be of little assistance to you ; you cannot diagnose broncho-pneumonia with certainty from simple bronchitis by physical signs ; you have always to consider the general symptoms. If there is high fever lasting more than two or three days, if there is much lividity and much depression, then you can be certain, no matter what the physical signs are, that you are dealing with the more serious affection—namely, broncho-pneumonia—and not with mere bronchitis. If there is one physical sign of assistance more than another, it is the development of fine crepitations towards the base ; and if in any case of bronchitis there are fine crepitations towards the base of one lung, you should be suspicious that you have to do with patches of broncho-pneumonia. The patches of broncho-pneumonia may be so small, and so deep down, and so enclosed by healthy lung, that there may be no dulness at all, and you must not expect always to get dulness when you percuss such a chest. But the more lightly you percuss it, the less likely you will be to miss such patches as exist, for you must remember that sometimes the patches are extremely thin; there may be only a little layer of broncho-pneumonia, and any heavy percussion will bring out the resonance of the underlying lung below that, and so you may miss the dulness.

DIAGNOSIS OF CROUPOUS FROM BRONCHO-PNEUMONIA.

The other disease from which you may have a difficulty in distinguishing it is croupous pneumonia, and it will be well for you to note some of the points which should guide you in the distinction between the two. Croupous pneumonia is always a primary disease, but broncho-pneumonia is very often secondary. Broncho-pneumonia is commonest in quite young children, in babies, whereas croupous pneumonia is not commonest in babies, but in children above two years of age. Broncho-pneumonia usually comes on more or less gradually, and the previous health has often been bad; whereas croupous pneumonia comes on suddenly, and usually in a child who has before been perfectly well. Broncho-pneumonia usually affects both lungs, whereas croupous pneumonia is generally unilateral, affecting one lung, or, much more likely, part only of one lung. In broncho-pneumonia the base of the lung is the part which is almost always involved, whereas in croupous pneumonia it is often apical. The course of broncho-pneumonia is ill-defined, and instead of ending by an abrupt crisis, it ends usually by lysis. In croupous pneumonia, on the other hand, the course is well-defined, and ends by crisis on the fifth to ninth day. The dulness in broncho-pneumonia is usually patchy and ill-defined, whereas in the croupous form it tends to follow sharply the outlines of the lobe. Another point of some value is that in broncho-pneumonia you get signs of general bronchitis, whereas in croupous pneumonia the rest of the lung seems healthy. These are the chief points to be attended to in making your diagnosis. The reason why it is so important to make the diagnosis is from the point of view of prognosis. Broncho-pneumonia is so fatal that about half the cases in hospital die, while, on the other hand, in croupous pneumonia recovery is the rule ; deaths are very exceptional. Holt, in his statistics, found something like 4 per cent. as the mortality of croupous pneumonia in young children, which is, of course, far lower than in grown-up people. There are probably two reasons for the better prognosis in croupous pneumonia in young children. One of them is the great reserve power of the heart, so that you do not easily get paralysis of the right ventricle from overdistension. The other reason probably is that children have usually very healthy kidneys, and there is nothing, I suppose, except alcoholism, which often goes with it, which makes the prognosis in pneumonia more unfavourable than damaged kidneys.

In spite of what I have said, I should be sorry if you went away with the impression that you should always be able to tell a case of croupous from one of broncho-pneumonia if you only pay attention to the points indicated above. That is certainly not the case ; often you will have great difficulty in distinguishing between them. For although broncho-pneumonia may begin in scattered patches, it it apt to become confluent, and to produce dulness over the whole of one lobe. Further, some cases of broncho-pneumonia present very much the same kind of temperature as one sees in true croupous pneumonia, and may even end by crisis. I have seen it stated that in such cases the pneumococcus is the exciting cause of the disease. Whether that is true or not I cannot tell you, but I think that it is probable that in future we shall learn to **classify cases of pneumonia** in children according to the particular organism which excites them rather than by their lobular or lobar distribution. From the point of view of prognosis, that would be a great advance, for there is reason to believe that cases which are due to the pneumococcus run a more favourable course than those which are of streptococcal origin.

In the **treatment of broncho-pneumonia** careful nursing is of the first importance. The child should be kept quiet and free from any excitement. He should be placed in an airy, well-ventilated room, and not in a tent. The bedclothes should not be too heavy, and the child's position in bed should be perpetually changed. Care should be taken not to overfeed. Even the best nurses are apt to fall into this error sometimes. The diet should consist of diluted milk and broths, and cold water may be given freely for thirst. Linseed poultices are sometimes useful if applied to the back, but they are apt to encumber respiration too much by their weight if placed on the front of the chest. Antiphlogistine is not open to this objection, and is useful if there be pain. If the temperature be running high, I have found cold compresses useful. They are prepared by wringing a towel out of water at the temperature of the room, wrapping it loosely round the chest, and then covering it with jaconet. The compress should be changed every three or four hours. Such applications are useful in several ways. They lower the temperature ; by making the child draw a deep breath when first applied, they tend to prevent collapse of parts of the lung, and they afterwards act as poultices.

Drug treatment is quite secondary in importance to the measures I have just described. Avoid the routine use of expectorants ; they are only useful when there is much coexisting bronchitis, and are then to be employed according to the same rules as in that disease. If cough is very teasing, small doses of paregoric are useful. Some also recommend belladonna, but I have not had much experience of it. Strychnine is of value as a general stimulant. It may be given in doses of 1 minim of the liquor every four hours to a child of one year. If there be much restlessness and a poor pulse, 15 to 30 minims of good brandy may be given in addition. In sleeplessness bromide of ammonium in 5 to 10 grain doses is probably the best remedy, but veronal may be used instead. If there be much cyanosis, oxygen inhalation should be tried, but too much should not be expected of it.

When the right heart is overdistended and the jugular veins very full and pulsating, I have often done venesection from the jugular with distinct benefit. A few leeches along the costal margin may be used instead if there is a prejudice against venesection, but they are not so effective.

During convalescence tonics and good feeding are necessary, and care should be taken to promote complete resolution of the affected parts of the lung. Change of air is often the most potent means of securing this end.

In concluding this subject, I may quote to you as a good summary of things to avoid an American doctor's* 'prescription for killing a baby with pneumonia':

'Crib in far corner of room with canopy over it. Steam-kettle; gas-stove (leaky tubing); room at 80° F. Many gas-jets burning. Friends in the room, also the pug-dog. Chest tightly enveloped in waistcoat poultice. If child's temperature is 105° F., make a poultice thick, hot, and tight. Blanket the windows; shut the doors. If these do not do it, give coal-tar antipyretics, and wait.'

PLEURAL EFFUSION.

Pleural effusions are not uncommon in children, and are apt to be overlooked. Here, again, physical signs are of doubtful value. There will be dulness over the effusion, but this need not be very intense; and not uncommonly, owing probably to the great conductivity of the child's chest, which I have already spoken of, the breath-sounds are preserved over the affected area, and may even have a feebly bronchial character. Hence, as you will readily understand, pleural effusion is not always easy to diagnose from consolidated lung, and sometimes you can only make sure of its existence by exploration. You need have the less hesitation in having recourse to this aid if you remember that in any case it is practically impossible to decide between clear fluid and pus without the use of the needle. The general symptoms will not help you, for clear effusion is often attended by a greater degree of fever than empyema; and, indeed, the symptoms of the latter in young children are often curiously slight. Even the presence or absence of a leucocytosis is no certain guide. It may be well for you to remember, however, that below the age of three clear effusions are rare, whereas empyema is comparatively common.

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* Dr. W. P. Northrup, *Medical Record*, N.Y., 1905, lxvii., p. 253.

The **treatment of pleural effusion** I need not describe, as it must be conducted on exactly the same lines as in grown-up people.

TUBERCULOSIS.

I have already referred to pulmonary tuberculosis in childhood, but I would again emphasize the fact that *phthisis as it occurs in the adult is practically unknown in children below the age of puberty.* It is true that phthisis is constantly diagnosed in children, because a slight cough is present, and the child is wasting, but the vast majority of such cases are really examples of chronic dyspepsia, with a throat cough. Patches of crepitation are also met with sometimes in the lungs, which, when they occur in the upper lobes, may closely simulate phthisis; but they clear up completely, and are apparently due to local areas of catarrh. The condition, however, which is perhaps oftenest mistaken for phthisis in the child is—

PULMONARY FIBROSIS.

This is relatively common, especially in patients of the hospital class, but it is very rarely tuberculous. In the great majority the condition dates from an attack of broncho-pneumonia in very early life, which has never properly cleared up, but has led to a condition of chronic interstitial inflammation, sometimes attended by dilatation of the bronchi and the formation of bronchiectatic cavities. The physical signs consist in dulness, usually over the lower part of the lung, with weak breath-sounds and crepitation. Here and there cavernous breathing and the occurrence of whispering pectoriloquy may indicate the existence of a cavity, and if the right base be affected, the heart may be displaced towards that side.

The symptoms are often surprisingly slight. The child may look fairly well—sometimes even blooming—but there is usually more or less cough, which varies greatly in severity at different times, and which may be attended by profuse expectoration, which is occasionally, though rarely, offensive. Attacks of intercurrent bronchitis or of localized pneumonia round the dilated tubes are common, and at these times more or less fever develops.

When the condition has lasted some time, the chest may become deformed and the fingers clubbed. The ultimate fate of these cases is uncertain. Many of them certainly seem to live a long time, but they may succumb to an intercurrent bronchitis or pneumonia, or to an attack of hæmoptysis. More rarely, secondary complications set in, such as cerebral abscess, and a certain number die in adolescence of right-heart failure, much as a case of emphysema in the adult does. In a small number tuberculosis becomes secondarily grafted on the original infection, and ultimately proves fatal.

The treatment of these cases is not satisfactory. If possible, one should guard them against further attacks of bronchitis by sending them to live in a warm, dry climate. When this is impossible—as it usually is—one should do all one can to maintain the general health at as high a level as possible, on much the same lines as in cases of asthma. Cod-liver oil is useful during the winter months. If there is much bronchiectasis, it is advisable to empty the tubes each morning by 'inverting' the child, and the administration of such 'pulmonary antiseptics' as creosote and guaiacol often may help. Garlic, also, is a useful drug, and children usually take it readily enough. It may be given along with cod-liver oil—*e.g.*:

Emuls. ol. morrh. }
Syr. allii acet. } .. .. .. āā ʒi.
Thrice daily after food.

I have tried vaccines in these cases without any success, the difficulty being to find the organism which is responsible for the condition. There is usually either no sputum to be obtained at all, or, if there is, it swarms with all sorts of bacteria.

When all is said and done, one cannot do much for these unfortunate patients, and therefore it is all the more important to prevent the development of the condition to start with, which can be done by insuring complete convalescence after every attack of broncho-pneumonia, no matter how slight.

LECTURE XVIII

SOME FUNCTIONAL NERVOUS DISEASES OF CHILDHOOD

GENTLEMEN,—In this and the succeeding lecture I propose to study with you some of the functional nervous disorders of childhood, which make up a group of diseases of great pathological interest and much practical importance. When you go into practice you will be struck by the frequency with which functional disease of the nervous system exhibits itself in your younger patients. One great reason for this, no doubt, is the comparative **instability of the nervous system** in early life. It is a nervous system which is, so to speak, still in the making. It has not yet settled down into the stereotyped form characteristic of mature life. The higher centres are as yet imperfectly developed, the lower incompletely controlled, and the paths of nervous impulses not clearly laid down. Hence abnormal forms of nervous action, manifesting themselves chiefly in defective co-ordination, spasmodic muscular contraction, and unchecked reflex excitability, readily become established. Several factors may come into play in rendering such abnormal action easier. First of these is a **nervous heredity.** Some babies are obviously 'neurotic' from the moment of their birth. They are easily frightened, and start and tremble at any sudden noise or unaccustomed sight. Such children, you can readily believe, will easily become the subjects of functional disease of the nervous system. But more important, because more frequent than a neuropathic heredity, are two factors which by universal consent are admitted to play a large part in the production of the group of diseases at present under consideration

These are in young children *rickets*, and in older children *rheumatism*. **Rickets and rheumatism** are the predisposers *par excellence* to all forms of functional nervous disease. Why should rickets render the children who are the subjects of it more liable to suffer from nervous trouble? I think it is probably to be attributed to the fact that rickets is, after all, only a form of malnutrition, and all forms of malnutrition tend to affect the highest centres first; hence the cortex is weakened, and its control over the lower centres diminished. How rheumatism predisposes to these diseases I do not know, but one knows as a clinical fact that rheumatic children are much more subject to functional nervous disorders of all sorts than those who are not similarly affected. I would take chorea as an extreme example of that tendency. Chorea is only a functional disorder of the brain, of some part of the cerebral cortex, and it is almost invariably associated with rheumatism. Now, chorea is a functional disorder which you are not likely to forget, because it is a dramatic disease and advertises its presence; but I want you to remember that what is true of chorea is true also of other less conspicuous nervous disturbances—namely, that they are specially prone to occur in children of rheumatic tendency.

Another point I want to insist upon before I proceed to deal with the different neuroses in detail is the question of **inco-ordination.** When you study those nervous diseases more closely you will find that what you have to deal with in nearly every one of them is an inco-ordinate action of the nerve-centres. It need not be surprising to you that a lack of co-ordination forms the basis of most of these neuroses, because, as you know, a baby is to a large extent an inco-ordinate machine; at all events, it has not yet learnt to harmonize many of its movements properly. A young child has not yet learnt to co-ordinate and control the action of the expelling and sphincter muscles of the bladder and rectum; nor has he learnt to control properly the muscles which hold up the head. And not even by the time he is a year old has he acquired sufficient co-ordinating power to enable him to walk. So you need not be surprised that a mechanism which has been but recently and imperfectly acquired should be readily thrown out of gear, and that inco-ordination should be a striking feature of most forms of functional nervous disease in childhood.

FACIAL IRRITABILITY.

I want now to pass to the study of some of those nervous disorders in detail. And first of all I wish to mention *facial irritability*, or Chvostek's sign. This is not a disease, but a symptom. It is a danger-signal by which you recognize the tendency on the part of the child to suffer from more serious forms of nervous disorder. Facial irritability is so called because when you tap over the facial nerve, below the malar bone, you get a contraction of the facial muscles. The phenomenon is not peculiar to the face, for if you tap almost any exposed nerve you get a similar muscular contraction. In other words, there is an exaggerated excitability of the nerves to mechanical stimuli as the basis of Chvostek's sign. If that sign is present you know that the nervous system is in an irritable and unstable condition, and will be prone to be affected by convulsive and spasmodic disorders.

LARYNGISMUS STRIDULUS.

Let us take next *laryngismus stridulus*. That is a serious and sometimes fatal form of nervous disease, which consists essentially in an inco-ordination of the muscles of the larynx. It is due to a spasmodic closure of the glottis, rendering the child for the time being incapable of breathing. Here you will almost invariably find that the [illegible] ing from rickets, that great predisposer to [illegible] You will get the history that from time to time [illegible] tops breathing, throws back the head, [illegible] the face, and then suddenly emits a crowing sound, after which he may cry a little as if frightened, but soon returns to his toys. There is here, then, a period of apnœa—that is to say, cessation of breathing—followed by a crowing inspiration; and these are the characteristic signs of laryngismus stridulus. These attacks will come on more in the night and in the early morning than at any other part the twenty-four hours, and they may easily be excited by any external irritation, by laughing, tickling of the throat, a draught of air blowing across the child's face when asleep, or by any emotional disturbance. At first the attacks occur only at rare intervals, but by-and-by they become more frequent, so that the child may have a great many seizures in the course of the day. What are you liable to mistake laryngismus for? First of all, you might mistake it for *congenital laryngeal stridor*. I shall speak of that condition more fully soon. Meantime I will only say that congenital laryngeal stridor dates from birth, whereas laryngismus stridulus usually affects children who are about eighteen months old. So that proper attention to the history alone in these cases will keep you right in the matter of diagnosis between those two conditions. Next you [illegible] mistake it for *croup*, the catarrhal laryngeal spasm of which I have already spoken; but croup attacks last for several hours, whereas laryngismus stridulus is almost momentary. Next, you might conceivably mistake it for *whooping-cough*; but whooping-cough is characterized by a series of short expirations followed by a whooping inspiration, whereas laryngismus stridulus is characterized by a period of apnœa followed by inspiration.

Laryngismus may be a serious disease. [illegible] I have said children sometimes die in an attack, [illegible] not be too confident in your **prognosis**. The story is told of a consultant who was summoned to see a case of this disease. He said there need be no alarm, as these children practically always got well; but as he was going out of the house he was called back to find that the child was already dead. We now come to **treatment**. First of all, how are you to treat an attack when it is in progress? What you have to do is to try to promote the taking of an inspiration, and this you can perhaps best do by dashing cold water on the child's face, or by tickling the fauces. If the spasm lasts an alarmingly long time, give a whiff of chloroform if you are able to do it yourself, or if there is some responsible person to whom you can entrust the administration. That will generally cause the spasm of the laryngeal muscle to pass off and re-establish the respiration. The application of a hot sponge over the larynx is also of use. Next, how are you to ward off future attacks ? A great deal can be done by keeping the child quiet. It must not be annoyed in any way ; it must not, as someone has said, be 'tickled, tossed, or teased,' because emotional disturbance and physical stimulation are exciting causes of attacks. It is well, also, to calm down the nervous system. The sedatives which you employ—and I shall have occasion to speak of them repeatedly—are bromides, chloral, and antipyrin. These three drugs are very useful in all the functional nervous disorders of childhood, and children stand all of them well, particularly chloral, which you can always give boldly. Seeing that rickets is the great predisposer to those attacks, I need hardly tell you how important it is to treat the rickets. That is never to be neglected. It is absolutely necessary to recognize the rickety element and to treat it by a change of diet, by cod-liver oil, iron, and so on ; and it is only in order to give those measures time to take effect that you meantime give bromides and chloral.

CONGENITAL LARYNGEAL STRIDOR.

I wish next to consider briefly the condition called *congenital laryngeal stridor*, which I told you might be mistaken for laryngismus stridulus. Congenital laryngeal stridor is a disease the **pathology** of which is still under dispute. It is believed by some to be due, like laryngismus stridulus, to an inco-ordination in the larynx. According to this view, it might be described as a stammer of respiration. When one makes an inspiratory effort, if the muscles are properly co-ordinated, the vocal cords fly apart to allow the air to pass in. And those who hold this theory maintain that in congenital laryngeal spasm this does not happen. The child raises his chest, but does not at the same moment open the larynx, and it is only a second or fraction of a second, later that the cords fly apart, and so there is a little catch as the air comes in. The other view—which is held by Dr. Lack, of this hospital, amongst others—is that you have to deal in congenital laryngeal stridor with a malfor-

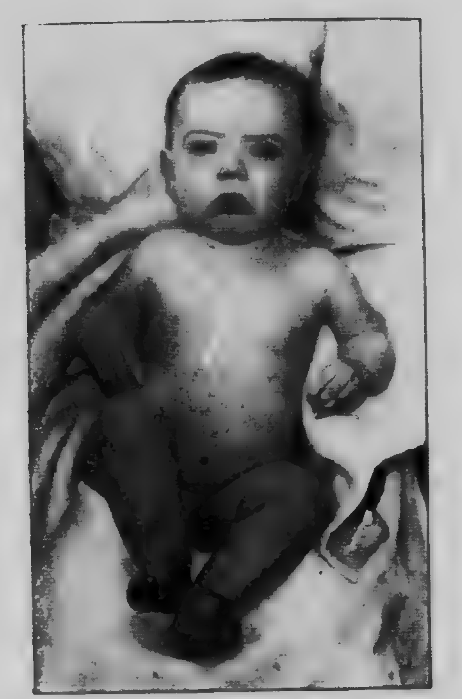

FIG. 27.

TETANY, SHOWING CHARACTERISTIC POSITION OF HANDS AND FEET.

mation of the larynx, consisting in a curving in of the epiglottis and flabbiness of the false vocal cords, which allows the larynx to fall in upon itself during inspiration, and that it is in that way that the symptoms are produced.* I shall not attempt to decide between these two views, but would say that it is admitted by all parties that the disease is congenital, and that towards the end of the first year of life it tends to disappear quite spontaneously. So far as I know it has never proved fatal. What are its **symptoms?** The child will be brought to you when quite a young infant, usually only a few weeks old, with the complaint that it makes a curious noise on breathing ; and you will be able to hear a slight crowing or purring going on in the throat, similar to the noise which you may hear a cat make. This sound goes on more or less continuously, but is apt to be louder if the child gets excited or agitated. During sleep the noise is almost inaudible. Laryngismus stridulus, on the other hand, is not congenital, and it is essentially discontinuous. One other thing which you may mistake it for is adenoids in their congenital form, but of this I shall have occasion to speak again (Lecture XXIX.). Inasmuch as congenital laryngeal stridor tends to disappear towards the end of the first year, it is not necessary to pursue any active **treatment.** I cannot satisfy myself that drugs have any effect upon it.

TETANY.

The next functional disorder of infants is the condition termed *tetany*, or *carpopedal spasm*, a name which describes the leading feature of the disease—namely, a spasmodic contraction of the flexor muscles in the front of the forearm and the back of the leg. You find the wrist flexed, and, as the interossei also participate in the spasm, there is flexion of the first phalanx and extension of the other two. In addition the thumb is often tucked into the palm, so that the hand assumes a conical shape, which has been described as the 'obstetric position,' because that is the position in which the obstetrician puts his hand when inserting it into the vagina. You may also find a degree of œdema on the dorsum of the hand, and that the skin looks pink or purple and somewhat glazed, and that on handling it it seems to be tender. What this is due to I do not know. It may be produced mechanically by the continuous spasmodic contraction of the muscles interfering with the proper return of the blood. The position of the feet resembles that of the hands. The toes are flexed and curled into the soles, the heels drawn up, and the dorsum of the foot is often somewhat puffy. If you examine the electrical reaction of the affected muscles, you find that their excitability is increased, and the anodal closing contraction is greater than the cathodal.

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* **See note at end of Lecture.**

The **causes** of this condition are two in number. In children of about eighteen months old it is very commonly associated with rickets. In younger infants you will find it coming on after an attack of diarrhœa. What the cause of the spasm is is not known. Sometimes you meet with a similar disease in grown-up people. It is one of the curious and rare complications of dilated stomach. It is assumed that in such a case you have to do with chronic poisoning, the result of the absorption of toxic material from the decomposing contents of the stomach. Arguing from that, some people say that tetany in young infants is produced in a similar way. It may be so, and its association with diarrhœa is suggestive ; but there is no proof of it. Tetany is not a fatal disease, but it is a painful one. You must first of all treat the rickets, if there be any, and, whilst your anti-rachitic treatment is taking effect, you must administer nerve sedatives. And of those I would recommend the bromides and chloral in preference to antipyrin. Hot baths are also useful, for they tend to relax the spasm when it is very severe.

HEAD NODDING.

Lastly, I want to speak of a disease which is of considerable interest and rarity, and that is *head nodding.* This condition of head nodding has several synonyms. Sometimes it is called spasmus nutans, sometimes gyrospasm, and sometimes it is spoken of as simply rotatory head spasm, or head jerking, or head rotation. Either head nodding or spasmus nutans is the best term to use. In it you have to deal with slight continuous movements of the head, which tend to occur in young children who are usually about a year old or a little older. When the child is brought to you the mother points out that the head is kept moving. Sometimes the movement is up and down, sometimes it is from side to side—a sort of pendulum movement—and sometimes a combination of those forms in different degrees. The essential point is a slight more or less continuous and apparently involuntary movement of the head. When you come to examine these cases more carefully you will find that a majority of them suffer from **nystagmus** also. The nystagmus may be from side to side, or it may be vertical or of the rotatory form, or it may be a combination of these varieties. And you may find in such a case a form of nystagmus which you do not find anywhere else—that is to say, a form in which the eyes tend to jerk towards one another and then to fly apart. In most cases of nystagmus, as you probably know, the axes of the eyes remain parallel, but to this form the term 'convergent nystagmus' is applied.

Here, again, it is believed you have to deal with an incoordination of the eye and head movements. The young child has just learnt to co-ordinate the movements of the eyes and head; he is just learning to turn his head in the direction in which he wants to look, and a very slight disorder may temporarily derange that co-ordinating mechanism. It has been compared to miner's nystagmus for a reason which I shall tell you of immediately. The interesting point which connects the movements of the eyes and those of the head is that the movements of the head tend specially to come on when the child tries to fix anything with his eyes. If you persuade the child to look steadfastly at you, you will find that the nystagmus increases and the head commences to nod. If the nystagmus is present in one eye only, the movements sometimes cease when that eye is covered up. This condition tends to occur with marked frequency at one part of the year—namely, in mid-winter. On a chart on the wall there are set out all the collected cases of head nodding which are on record,* and you will observe that the maximum frequency is in the month of January. In the other months you will see there are hardly any cases at all. Why is head nodding almost confined to this particular part of the year? An explanation which has been advanced is that it is due to the absence of sunshine at this time. And some of you who saw a recent case of the disease in the outpatient room will remember that the mother admitted that the child lived in a very dark room on the ground-floor; and it is under such conditions that the disease most commonly appears. That is why people have compared this form of nystagmus with that known as miner's nystagmus, which is due to the constant strain of using the eyes in comparative darkness. Now, although that explanation is ingenious, and has much to commend it, it is not universally accepted, and in many cases it is impossible to find that the children who are the subjects of the condition have lived in dark houses; some of my cases have occurred in the suburbs or in the country, where there is as much light as can be met with in this country in the winter.

That brings us to speak of the **diagnosis.** The only condition for which you are likely to mistake head nodding is *salaam convulsions*, which consist essentially of swaying or nodding movements of the body, which appear to be epileptic in their nature, one of the forms of *petit mal*. But head nodding is associated with nystagmus, and occurs particularly at one time of the year, and tends always to pass off after a short time. There are certain automatic movements which idiots are apt to make which you may also mistake for head nodding, such as swaying movements, banging of the head, or knocking of it against the pillow; but all these movements are much more gross, and are larger in their range, than the typical head nodding.

The **prognosis** of this disease is perfectly good. If you leave it alone it will always get well in time. Therefore you can reassure the mother. Sometimes, however, the nystagmus seems to persist after the head nodding has passed off; and it is possible that some cases of nystagmus which are alleged to have begun in childhood, and for which no cause is assigned, have been cases of nystagmus with head nodding in which the eye movements have persisted.

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* From a paper by Dr. John Thomson in Jacobi's 'Festschrift,' New York, 1900, p. 282.

The **treatment** which is most likely to cut short the movements and produce improvement is the administration of small doses of antipyrin—1 grain for every year of life. This can be given two or three times in the day. I think I have seen this drug effect a distinct and decided improvement in the movements, rendering their occurrence less frequent and less marked; but the nystagmus seems to be more difficult to control.

Somewhat akin to head nodding, but clearly to be distinguished from it, is the condition known as **head banging.** This affection is characterized by a violent banging of the head against any solid object that comes handy—the floor, the wall, the side of the cot, the pillow, etc. It usually occurs in children of about two years of age, and the violence of the bangs may be such as seriously to alarm the mother for the child's safety. I have never known one, however, do itself any real injury, though occasionally a little bruising may result. What satisfaction the child finds in this exercise it is difficult to see, but the probability is that it is performed in order to relieve some uncomfortable sensation in the head, such as earache or the pain of teething. No special treatment is required, though it is well to see that the child does not do himself any injury. A course of bromides and antipyrin may be tried in bad cases.

NOTE.—Paterson (*Brit. Med. Journ.*, 1906, ii. 1447), from careful observation of several cases during life, concludes that there is no evidence of any inco-ordination of respiration and no malformation of the larynx in congenital laryngeal stridor beyond exaggeration of the natural incurving of the infantile epiglottis. The stridor is produced at the entrance of the larynx by the vibration of the soft structures on the posterior wall.

LECTURE XIX

SOME FUNCTIONAL NERVOUS DISEASES OF CHILDHOOD—*Continued*

GENTLEMEN,—In the last lecture we began the consideration of a group of functional nervous disorders in children, the essential nature of which, we saw, was that they consist in an inco-ordination of movement. We saw, further, that there were several reasons why children are particularly subject to such inco-ordinate action of muscles, one of the chief being that, in the case of young children, at any rate, co-ordination is a recently acquired function of the nervous system, and therefore is one which is very easily lost under adverse conditions. We saw that amongst such adverse conditions must be placed many diseases of nutrition, rickets being one of the most conspicuous. In addition, it was pointed out that in older children rheumatism plays a part in the production of functional nervous disease, and that the factor of heredity can never be altogether ignored. We reviewed some examples of functional nervous diseases, taking up first the diseases of the larynx — congenital laryngeal stridor and laryngismus stridulus — and then we passed on to tetany and head nodding. I wish now to conclude this subject with you, beginning with the important question of convulsions.

CONVULSIONS.

You will meet with convulsions in children very frequently in your practice, and they are very alarming to the parents of the child. And, further, from the point of view of the medical man, their exact causation is often difficult to diagnose. I want first of all to warn you that mothers and nurses will call by the name of convulsions many things which really have no right to be so called. They will tell you, for instance, that the child is suffering from 'inward convulsions,' or 'inward fits,' or 'screaming convulsions.' And they may mislead you into thinking that you have to do with a case of genuine convulsive disease. Many children under the irritation of teething or colic, or under emotional disturbance, exhibit twitchings of muscles, particularly muscles of the face, which the parents are apt to suppose are true convulsions. But you are not to regard mere quiverings of eyelids and screwing up of the mouth, or violent fits of screaming, as genuinely conclusive, though they may be of value to you as warnings that a tendency to convulsions exists.

You will find it convenient to divide genuine convulsions into two main groups—first, those which are *idiopathic;* and, secondly, those which are *symptomatic.* By **idiopathic convulsions** one means those for which one can find no cause, which arise in a child who is apparently in fairly good health, and which may pass off after a time, or which not infrequently become established, so that the child suffers from fits from that time onwards at varying intervals. There can be no doubt that a considerable number of those cases are examples of genuine epilepsy. Epilepsy may certainly begin in early life, so that when you are called to a child who is suffering from violent convulsions for which you can find no cause, you must bear in mind that you may be dealing with the first stage of that disease. Sir William Gowers puts the proportion of cases of epilepsy beginning as convulsions in infancy at 10 per cent., and some authors give an even higher figure.

Far more important, because more common, are cases of the symptomatic group. In the first place, you may have **symptomatic convulsions** as the equivalent of a *rigor* in a grown-up person. Many young children, instead of having a rigor at the outset of an acute illness, have actually a general convulsion. This may happen at the commencement of pneumonia, or scarlet fever, or any of the other acute specific fevers, where the temperature runs up rapidly during the first few hours of the illness. Secondly, symptomatic convulsions may be an indication of **organic disease of the brain.** They may occur in meningitis in all its forms, or they may be met with in cases of cerebral tumour, or with thrombosis of cerebral veins. Indeed, in almost any serious intracranial disease in children you may find convulsions as one of the symptoms. Thirdly, convulsions may be of **reflex** character or origin, an expression of the reflex irritation which arises from such things as the cutting of teeth or the existence of colic or phimosis. The reflex irritation which results from these causes may, as it were, 'fire off' convulsions from the cerebral cortex. There is another group which is **toxic** in origin. Uræmic convulsions are a typical example of this group ; and I think it is not unlikely that some of the cases of convulsions which you meet with in the course of acute diarrhœa can also be attributed to toxic causes. Lastly, there are the convulsions which are the result of **asphyxia.** Such are those which you may sometimes meet with in the course of pertussis. Towards the end of any illness also convulsions may come on and be, so to speak, one of the modes of dying. These also are probably due to failure of respiration or of the heart, leading to cyanosis and congestion of the brain.

Those are the chief groups into which you can divide cases of convulsions ; and when you are called to a case you have to decide, as far as you can, to which one of these classes it belongs.

You will find that, just as in many of the other functional neuroses we spoke of last time, so here **rickets** is the great predisposing factor. For instance, the cutting of teeth in a child in good health may not set up convulsions, but if the child be rickety, such irritation may be a determining cause of spasmodic discharges from the cortex of the badly-nourished brain. The same is true of the other causes of which I have spoken. Many of them are insufficient by themselves to produce convulsions in an ordinary child, but where rickets has produced nervous instability they may suffice as determining causes. You can sometimes perceive that convulsions are about to come on by certain warning signs. One of these is the development of the facial irritability of which we spoke last time, and which we saw to be an indication of undue irritability of the nervous system. Another will be slight twitching movements of the face and extremities. If these twitchings are seen in a child the subject of rickets you ought to anticipate that the child may become affected by convulsions, and do what you can to ward them off. Sudden flushings or attacks of coldness are also occasional premonitory signs.

When you consider the **prognosis of convulsions** you have to take into account what the immediate chances are, and also what is likely to be the remote outcome of the seizure. Children do not often die in a convulsion, unless the convulsions are themselves merely the final scene in an already mortal complaint—that is to say, unless they are simply a symptom of the terminal asphyxia. Convulsions as a disease are not immediately fatal, unless when very severe and repeated and in quite young infants. You have to remember, however, that in a considerable proportion of the cases the convulsions may persist in the form of epilepsy; and you have, further, to remember that nearly all, or quite a large number, of the children who suffer from convulsions manifest in later life nervous disorders of different sorts. Of about eighty-five cases followed up by Dr. Coutts and traced into adult life it was found that eleven suffered from major epilepsy, four from *petit mal*, five exhibited somnambulism, four developed melancholia, seven had chorea, and nine suffered from migraine. So you see in what a large proportion of those cases the later history of the patient is marked by further nervous disturbance. In addition to this, convulsions, especially if frequently repeated, may cause permanent mental impairment, and they often leave behind, too, traces of cerebral damage in the form of stammering or a squint.

With regard to **treatment,** your immediate line of action when called to a child who is at that moment the subject of a convulsion must be the same, no matter what the cause of the attack may be. First of all, you lay the child down raising the head somewhat, and loosen the clothing just as you do in a case of epilepsy, so that there is no hampering of respiration. And it is a good thing, unless you have been anticipated by the mother or nurse, to put the child into a warm bath, though this is perhaps most efficacious in those cases which are the result of colic. Next you should give sedatives by the mouth, or, if you are unable to get the child to swallow, by the rectum. Chloral is the most efficient, 3 or 5 grains by the rectum to a child six months old. If you fail, and the convulsions are very serious and still persist, and the friends are getting alarmed, there is one resource which is open to you, and which I think is practically certain to be successful—namely, the inhalation of chloroform. A few whiffs of chloroform given on a handkerchief will cause the convulsions to cease by paralyzing the brain cortex. In bad cases in which the child passes from one fit into another you may require to give morphia hypodermically, but it must be used with caution.* Amyl nitrite also is sometimes of service. Such will be your immediate treatment in all cases. Next you have to consider how you are to prevent the recurrence of the attacks In order to do that you must treat the rickets, if there be any. You must make a radical alteration in the child's diet and administer cod-liver oil. But occasionally, and until these measures have time to take effect, you may have to diminish the excitability of the nerve centres by sedatives. Bromides and chloral will be the drugs which you will use. I would remind you again here how well young children stand chloral; it is a drug which you may give pretty freely without risk. And in those cases it is to be preferred to bromide, as being more efficient in controlling the convulsive tendency. It may be necessary, of course, to attend to the child's digestion if there be colic, and that may involve the alteration of the diet in directions I have laid down in a previous lecture.

Before leaving the subject of convulsions I wish to mention a condition which resembles them somewhat, and which you will not find described in your text-books. The following extract from the letter of a doctor who wrote to consult me about it in one of his own children will give you a good idea of what I mean :

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* The dose is about $\frac{1}{50}$ grain for a child of six months.

> '. . . The patient is my third child, a boy of two. He has always been healthy, and there are no signs of rickets or other constitutional disease. Ever since he was quite small he has been in the habit "of holding his breath." The condition has become worse lately, and during the last few days he has had several such attacks. The child begins to cry, respiration is suspended, he gets purple in the face, and then becomes unconscious. The spasm is then relieved, and he begins to breathe, but twice he has had very marked twitching of the face muscles, which, however, soon passed off. He remains quite inert and irresponsive for fifteen to twenty minutes, when he suddenly recovers, and, except for being upset and white-looking, is himself again. There is *no* crowing respiration.'

I have seen several cases of this sort, all of them in boys below the age of five, but so far as I am aware there is no scientific name for the condition, although I believe it is known in some parts of the country as the **kinks.** It consists, apparently, in a spasm of the respiratory muscles, which differs from the spasm of laryngismus in that the chest is fixed in the position of inspiration instead of expiration—hence the absence of a 'crow' when the spasm passes off. The subjects of this neurosis are usually excitable, self-willed children, and the spasm tends to come on whenever they get in a temper or are crossed in any way. Although the attacks may be very alarming, I have never known them lead to any fatality.

The best treatment is to dash some cold water in the patient's face so soon as an attack is seen to be coming on ; this will usually stop it at once. In addition, such children should be guarded from all excitement, be firmly managed, and, if necessary, be put through a course of bromides.

NIGHT TERRORS.

I now pass to another extremely important group of nervous disorders, those which go by the name of *night terrors*, or *pavor nocturnus*. Night terrors of varying severity occur with a considerable degree of frequency in children, and they are often extremely alarming to the friends. The usual history of a case is that a child, most commonly about the period of the second dentition, falls to sleep, and shortly afterwards wakes up in a violent state of terror ; he sits up in bed and shouts in an incoherent manner, perhaps only partially awake, and it may be a long time before the mother or the nurse succeeds in pacifying him. In those cases the cause of the fright is sometimes an hallucination of vision ; sometimes, on the other hand, it is probably merely a nightmare. It has been aptly said, I think by Dr. Coutts, that you may divide the cases into two groups—'those that see visions, and those that dream dreams.' Those who see visions are those who have hallucinations of sight ; those who dream dreams are those who have what is equivalent to a nightmare in the adult. Although that is probably a real distinction, you will find it extremely difficult to say to which of those two classes any given case belongs, because these children are often incapable, sometimes unwilling, to give an accurate description of their sensations during the attack. Sometimes they will tell you that they see frightening objects, shadows or dark shapes in the room, and in such cases, perhaps, they are the subjects of visual hallucinations. In some cases they can give you no coherent account of their sensations. These experiences may be repeated night after night, until the equilibrium of the nervous system is seriously upset, and the child begins to dread going to bed at all ; and then the friends get alarmed, and think that the child is becoming insane, and call in a doctor.

A very good description of the sensations of children who suffer from those attacks has been given by Charles Lamb in one of the 'Essays of Elia,' entitled 'Witches and other Night Fears.' Perhaps the passage is known to some of you. He says: 'I was dreadfully alive to nervous terrors. The night-time, solitude, and the dark, were my hell. The sufferings I endured in this nature would justify the expression. I never laid my head on my pillow, I suppose, from the fourth to the seventh or eighth year of my life [*i.e.*, about the time of his second dentition], so far as memory serves in things so long ago, without an assurance, which realized its own prophecy, of seeing some frightful spectre. . . . Parents do not know what they do when they leave tender babes alone to go to sleep in the dark. The feeling about for a friendly arm, the hoping for a familiar voice when they wake screaming and find none to soothe them—what a terrible shaking it is to their poor nerves! The keeping them up till midnight through candle-light and the unwholesome hours, as they are called, would, I am satisfied, in a medical point of view, prove the better caution.'

Now, gentlemen, night terrors may always be taken as evidence of ill-health in some form or other. They do not occur in perfectly sound children. And you will find that amongst the **predisposing causes** particularly at work are, in the first place, a neurotic or nervous heredity; secondly, you will find a rheumatic diathesis, as I have already stated. But, above all, and far more important than all, perhaps, is overpressure at school. One of the chief predisposing causes of night terrors is school work, and you will often in many cases find that children only suffer from this disorder during the school term. During the holidays they sleep undisturbed, but when they resume school they resume also their night terrors. Mothers often describe them as 'going over their sums in their heads all night,' arithmetic being apparently one of the chief things which exhaust and irritate the brain.

Of **exciting causes,** I would emphasize two which it is of the very first importance for you to be familiar with. One of them is gastro-intestinal disorder, especially *constipation*. The second is partial asphyxia from the presence of *adenoids* in the throat. These two causes are by far the most important for you to recognize from a practical point of view.

When I have mentioned to you the causes of night terrors, you see what the **treatment** should be. It must not be purely sedative. There are two things above all that you should do when called to a child who suffers from this complaint; one is to attend to the state of the throat, and the other is to administer aperients. If you do these you may leave almost everything else undone. Clear out adenoids when you find them, and give an aperient, no matter what the state of the bowels may be or is said to be. If you do that you will not go far wrong in the management of those cases. There are other accessory means which are of help. These children ought not to be frightened in any way, nor excited. They ought not to be allowed to sleep alone, they ought to have a night-light in the room, and ought not to be allowed to go to bed with a full stomach, because, as in nightmare in grown-up persons, so here, pressure of food in the stomach may be a cause of the condition. It may be well to remove the child from school for the time being, until the nervous system has recovered its equilibrium. It may be necessary to give sedatives, such as bromides and chloral, temporarily; but as far as possible you should dispense with these and direct your attention to putting the child into that state of health in which night terrors are impossible, and in order to do that, as I have said, you must pay attention to the state of the throat and stomach and bowels.

There is an analogous condition to night terrors which occurs in the day, called day terrors, or **pavor diurnus,** which is very much rarer. The child, in the middle of his games, may be seized with a fit of panic and rush to his mother, giving vent to inarticulate cries. The fit of terror may last for a few moments. These are no doubt to some extent analogous to the worst form of night terrors. They are probably cases where there has been some hallucination of some of the sense organs, and must be treated on the same lines as the night terrors of which I have spoken.

There are also various degrees of disturbance and restlessness at night, which cannot, perhaps, be fairly classified as night terrors. One of the mildest forms is **grinding of the teeth.** Such cases can be recognized by the fact that the teeth are bevelled down to the same level. At the other extreme is **somnambulism.** All such cases must be treated on the lines already laid down,.

NOCTURNAL INCONTINENCE.

The next neurosis I shall describe is enuresis, or *nocturnal incontinence.* I do not pretend to explain how it is that enuresis arises. The exact mechanism of the bladder is not fully understood, but for practical purposes you may

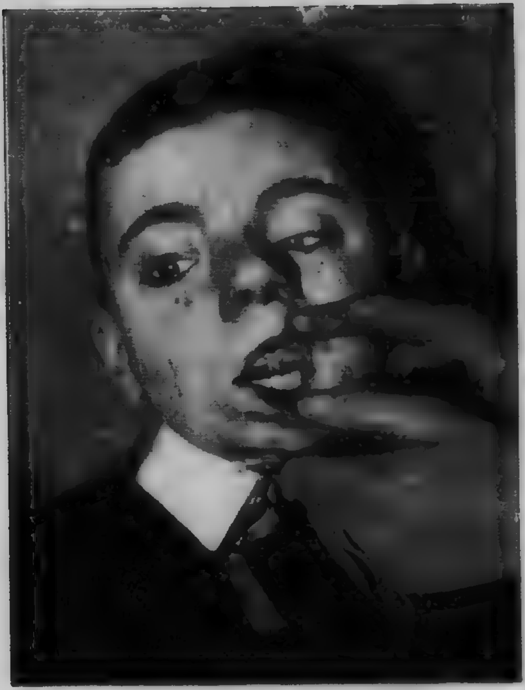


Fig. 28.

TEETH-GRINDING.

Note how the upper teeth are worn down to a level.

compare enuresis to the automatic action of the bladder which you find in many diseases of the spinal cord ; and probably it has a very similar explanation. Just as in diseases of the spinal cord, when the brain impulse is cut off by a lesion higher up, the bladder takes to emptying itself automatically when it is full, so in children the brain cortex has sometimes such an imperfect control over the lower centres that the bladder may take on spontaneous action. You may ask at what age the child ought to be able to control the bladder. I think you may say from three years old. You should not therefore talk of enuresis before that.

The **treatment** of the spontaneous passage of urine during the night must consist in the removal of the causes which lead to it. You will find it always well to inquire into the presence of worms, because the irritation of worms in the rectum seems to be a cause which may give rise to enuresis. You should also inquire in the case of boys about phimosis, or preputial adhesions ; these also seem to be one of the exciting causes. Further, you should see that these children do not drink much fluid before going to bed, that they sleep on a hard mattress, with few bedclothes ; and it is advisable to raise the end of the bed a little, so that the head is lower than the pelvis, and the stress or pressure of the urine tends to gravitate down into the fundus of the bladder instead of pressing on the more sensitive trigone. Here also you will find in a great many cases that partial asphyxia is the cause of the trouble. And adenoids, again, are by far the most common cause of the partial asphyxia. So that in every case of nocturnal incontinence which is brought before you you should make a point of examining the throat. Lastly, it is well to examine the condition of the urine, to see if it be acid to an undue extent. I think hyperacidity of the urine as a cause of nocturnal incontinence has been exaggerated. I do not think that it is a common cause. If, however, you find uric or oxalic acid crystals, it is well to give alkalies to make the urine less irritating. I have even seen calculus in the bladder mistaken for incontinence, from the irritation and frequent micturition to which it gave rise.

If you have employed all these measures—attended to the state of the urine, removed worms and adenoids if they were present, given directions that the child should not have fluid just before going to bed, and that he should sleep on a hard mattress and with few bedclothes—and if you still fail to cure the condition, what shall you do next? You have then to fall back upon drugs. There is one drug especially which is commonly used in the treatment of nocturnal incontinence, and that is belladonna; and there can be no doubt that it is a drug of extreme value if you use it in the proper way. So far as I know, it is the only drug which has a real controlling influence in the disease. There is only one rule to be remembered in regard to the **administration of belladonna,** and that is *to give it in large doses.* If you do that, you do not need to trouble about any other points. Begin with 10 minims of the tincture, given after each meal; try that for a week, and you will probably reduce the frequency of the incontinence at once. You should then mount up to 15 minims each dose, then to 25 minims, and then to 30 minims, and so on, until you get the incontinence entirely suppressed. I have no hesitation in going up to a drachm of the tincture, even of the new B.P. strength, after each meal—that is to say, three times a day. It is extraordinary how children will stand belladonna. It is one of the drugs which have far less influence on a child than on a grown-up person. There may possibly be a complaint of slight dimness of vision from paralysis of the eye muscles, or of dryness of the throat, during the administration; but I have not seen more than once or twice any general disturbance, such as a rash or slight delirium. As a rule, there are hardly any general toxic effects from large doses, whereas the local effect upon the enuresis is quite remarkable. Now, if you have got up to 40 minims of the tincture after each meal, and then the enuresis ceases, how long should you continue the drug? I think you should do so for at least a month in the full dose, and then discontinue gradually; climb down again in the matter of doses just as you climbed up. You will then find that in the majority of cases when you have stopped the medicine the habit has been broken, and the child may be regarded as cured. Other drugs are sometimes recommended as substitutes for belladonna. Ergot has some supporters, strychnine has some, and so has cantharides. I have tried all of them, I think, and I can only say that I am by no means certain that they are of much use. I should say that if you fail with belladonna you may regard yourself as at the end of your therapeutic resources so far as drugs are concerned.

I think you will find nocturnal incontinence of urine is much more easy to cure in boys than in girls. I do not know why that should be, but I think the most intractable cases one has to deal with are in girls. In such cases treatment by local applications of nitrate of silver to the urethra has recently been recommended, a strong solution being applied on more than one occasion. I have seen one case at least where this method effected considerable improvement, but my experience of the treatment has not been great. Still, it is one which it is well to have in reserve in the case of girls, for whom alone it is intended.

Finally, I would warn you that you must see that these children are not punished. It never does any good, or hardly ever, to do so. Very few cases are due to carelessness on the part of the child ; the disorder is due to a want of control of the lower centres by the higher, and the lack of control is not a voluntary one, but purely involuntary. It is a case in which the child cannot exert the will, and therefore it is not only useless, but it is extremely unfair and unkind to punish children for this unfortunate weakness. On the other hand, by carefully following out the treatment I have described, I think it will be your experience that in the majority of cases you can get the patient well.

Occasionally the incontinence **occurs by day only,** though that is exceptional, and the treatment is essentially the same as in the nocturnal form. I remember being consulted, for instance, about a girl nine years old, who had suffered from diurnal incontinence since an attack of measles three years previously. She was a very nervous child, but presented no other signs and the urine was quite normal. Under full doses of bromides and belladonna, the symptoms speedily disappeared.

Incontinence of Fæces is a rare condition which may occur either independently or in association with enuresis. Not long ago I had a case of it in a girl of five, who seemed otherwise in good health, but who had never been able to control the rectum. She recovered completely under belladonna and ergot.

HABIT SPASM.

Habit spasm, or tic, is the last of the functional neuroses of childhood which I shall describe, but it is by no means the least common. It consists in an oft-repeated and apparently purposeless movement usually affecting the face, and resulting in 'blinking,' 'grimacing,' 'sniffing,' or other such act. Movements affecting the limbs are also seen, the upper limbs being more often affected than the lower. Thus, there may be a constant shrugging of one shoulder or repeated snapping of the fingers. In severe cases there may even be involuntary exclamations—often of an improper sort.

The subjects of habit spasm are neurotic children, and, as in other functional neuroses, often exhibit a tendency to rheumatism. Overstrain at school, anæmia, and feeble general health, predispose to its occurrence. Local exciting causes should always be looked for. Amongst these are errors of refraction, adenoids, and carious teeth.

The diagnosis is usually easy, but you will be apt to mistake the condition for chorea. In typical habit spasm, however, only one group of muscles is affected, whereas in chorea that is very rare. There is no doubt, however, that the two disorders tend to pass into each other, and it may not be possible at first to distinguish a severe habit spasm from a case of mild or of 'residual' chorea.

The treatment to be adopted is much the same as that of the other functional neuroses—viz., attention to the general health, and avoidance of excitement and school-work, with plenty of exercise in the open air. If any of the local exciting causes already mentioned is present, it should be dealt with. Thus it may be necessary to order spectacles, or to attend to the teeth or throat. Removal of adenoids, however, should not be lightly undertaken, as the fright of the operation may aggravate the spasm. Gentle but persistent 'moral suasion' may do something, the child being constantly encouraged to try to control the movement. Anything like scolding, however, should be avoided. As regards drugs, I have found most benefit from a combination of bromides and arsenic, but valerian is also useful occasionally.

As a rule, the condition is recovered from in time, although improvement is very often slow, and sometimes the movements become stereotyped and persist into adult life. When all else fails, a complete change of surroundings and going to live amongst strangers may have a good effect.

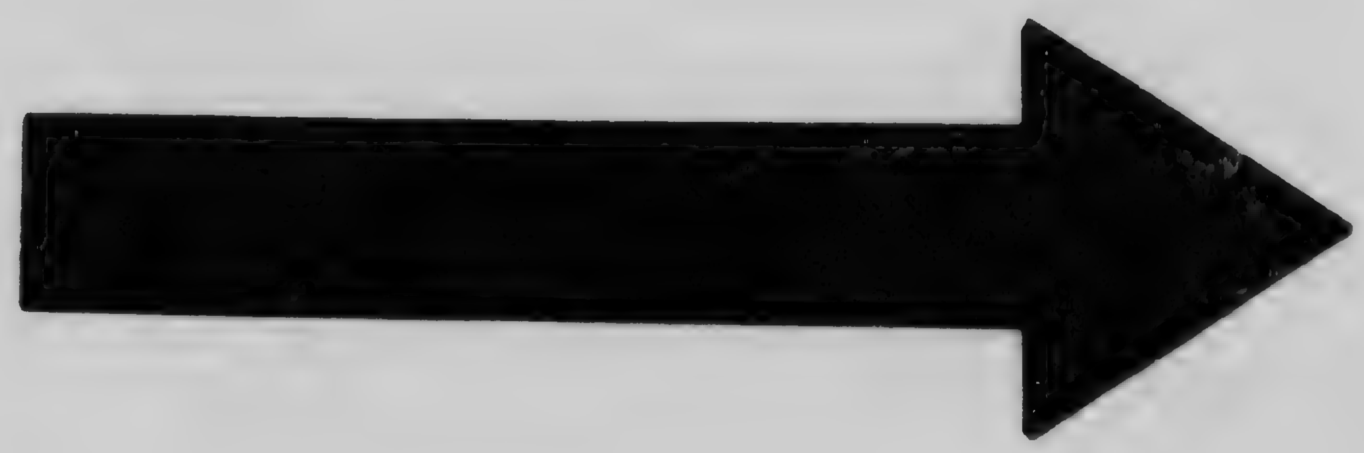
LECTURE XX

HYSTERIA IN CHILDHOOD

GENTLEMEN, My subject this afternoon is hysteria in childhood, and you might perhaps expect that at the outset I should define what I understand by the term 'hysteria.' But in that I am afraid that I must disappoint you, because, frankly, I do not know of any satisfactory definition. And if you attempt to penetrate into the inner meaning of 'hysteria,' you simply involve yourself in the fogs and bogs of a pseudo-psychology. That, at least, is the conclusion I have come to from a survey of the recent literature on the subject. I shall content myself, therefore, with a purely objective description based on my own clinical experience, and shall be satisfied if I convince you that hysteria is, indeed, a possibility to be reckoned with when one has to do with a case of obscure nervous disease in early life. Although I shall have many cases to relate, you must not make the mistake of supposing that hysteria is common in childhood. On the contrary, it is rare, and I doubt whether it can be recognized with certainty below the age of five. At the outset certain points may be mentioned in which hysteria in childhood differs from the same condition in the grown-up person. One is with regard to sex distribution. There is not the same disproportionate occurrence of hysteria in the female sex in children that there is in adults. Indeed, I think you will find when I come to describe cases that it is quite as common in boys as it is in girls. But the nearer you get to puberty the more does the tendency for the female sex to preponderate as subjects of the disease exist. Another difference which one notices between hysteria in children and in grown-up persons is that in children it tends to assume a simpler form. It is often what is cumbrously termed 'mono-symptomatic'; that is to say, there is only one hysterical manifestation present. In that hysteria simply obeys the rule which I have pointed out in these lectures before—namely, that all disease processes in children tend to be simpler than in grown-up persons. And to that hysteria is no exception. Another point characteristic of hysteria in the child is that many of the so-called stigmata, such as anæsthesia of the pharynx, contraction of the visual fields, pressure points, and so on, are either not met with at all, or only occur exceptionally. Why that should be I do not know, but it is a clinical fact which you must bear in mind.

With regard to the **etiology** of hysteria in children, I think one can recognize with a fair degree of frequency two kinds of causes—(1) predisposing, (2) exciting. A large number of hysterical children are disposed by inheritance to suffer from the condition. You will be surprised how often, when you are dealing with an hysterical child, you will find that it has an hysterical mother, or that it is the offspring of neurotic or highly emotional parents, and this predisposition has often been aggravated and fostered by the circumstances of the patient's environment. The child is apt to have been badly brought up, not in the sense of having been neglected—on the contrary, it has probably been coddled and spoilt—but it has not been brought up to exercise self-control, and very often in an emotional and exciting atmosphere.

Of the exciting causes of hysteria in childhood the commonest is injury of some sort, physical injury or trauma. I shall give you some concrete examples of this immediately. The other great exciting cause is what has been called 'psychical trauma'—namely, fright—and in a number of instances hysterical manifestations have followed upon this. That is all I can say definitely from my own experience about etiology, but in a great many cases you will be unable to find any cause for the condition at all.



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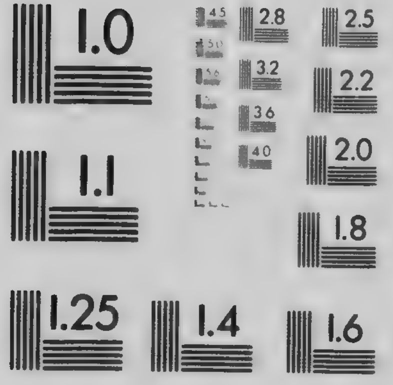

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MOTOR MANIFESTATIONS.

Hysteria may assume various forms, the commonest, perhaps, being affections of the motor system, which may show themselves as paralysis, fits, spasms, or tremors. And, first, let us speak of **hysterical paralysis.** Of the different forms of hysterical paralysis in children, monoplegias and paraplegias are the commonest. Hemiplegias are less frequent. The paralysis is often of sudden onset, and it usually assumes the flaccid or limp type. Hence the case is apt to be mistaken for one of infantile paralysis, as happened in the following example :

Case 1.—A boy, aged 12 years, was brought up from the country with a history of paralysis of the right arm, which had come on suddenly five months before. The paralysis had been regarded as due to poliomyelitis. On examination, there was complete flaccid paralysis of the right arm, but with only a moderate amount of wasting. There was anæsthesia of ‘ sleeve ’ distribution up to the shoulder. Otherwise he seemed normal. Under the application of faradism, accompanied by vigorous suggestion, he immediately recovered the use of the limb, and returned home next day cured.

I have not myself seen a case of hysterical paraplegia in a child, but those who are interested in the subject will find a very instructive example of it, which led to various errors in diagnosis, recorded by Dr. C. W. Chapman in the fifth volume of the Transactions of the Society for the Study of Disease in Children.

Hemiplegias are comparatively rare, and when they do occur the face usually escapes. The following is an example :

Case 2.—Susan C——, aged 12 years, got a severe fright at a fire two years ago, and had had several ‘ hysterical ’ fits since. After one of these lately she lost the use of the right arm and leg. The arm quickly recovered, but the right leg remained weak, the foot being bent in the equino-varus position, and dragged along as she walked.

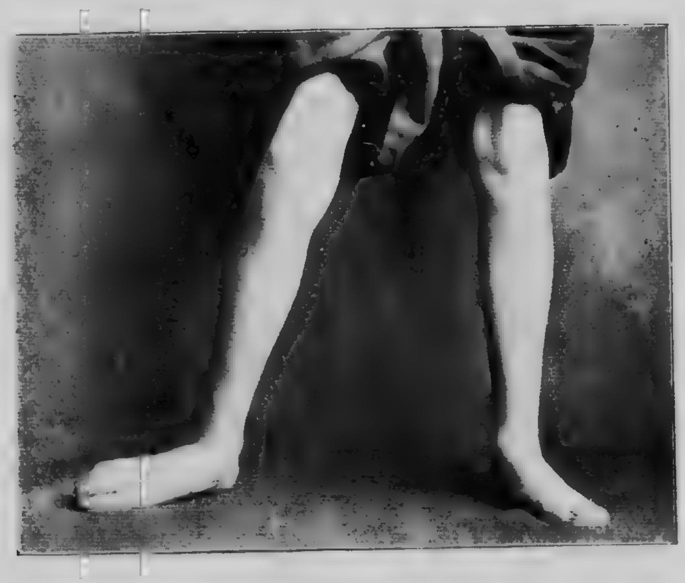

FIG. 29.

HYSTERICAL PARALYSIS IN RIGHT LEG, SHOWING HOW THE FOOT IS DRAGGED.

Examination showed anæsthesia over the whole of the right half of the body, with amblyopia in the right eye and considerable areas of analgesia in the leg. Otherwise examination was negative, but her mother said that she would sometimes refuse all food for two days on end.

She only came once or twice to the hospital, and her subsequent history is unknown.

The curious condition called 'hysterical astasia-abasia'—inability to stand or walk—seems also to occur sometimes in children, but not commonly. Usually I think it is left behind as the result of a condition which at the outset did really make standing or walking painful, and then, after that has passed off, the astasia-abasia persists, owing to the hysterical tendency. This seemed to happen in the following case :

Case 3.—Albert L——, aged 5 years, 'went off his legs' when eighteen months old (? result of scurvy), and had never walked since. He seemed quite healthy, there was no paralysis, and he kicked his legs about freely in bed. The knee-jerks were rather brisk, and the plantars gave a flexor response. At first, when one tried to make him stand, he simply screamed and doubled up his legs ; but after a few days of persuasion he stood up, and four days later was walking quite well.

There was a good deal of anorexia whilst he was in hospital, and it was difficult to induce him to take enough food.

This, I think, was a case which might be fairly described as hysterical astasia-abasia. It had started, I believe, owing to the tenderness of the limbs produced by scurvy, and afterwards the inability to walk was perpetuated in an hysterical form.

Passing from hysterical paralysis, we come to **hysterical fits.** They are, as you know, a very common manifestation of hysteria in the adult, the typical case being that of a young woman who gets an 'hysterical attack' in which she throws herself about, cries, laughs, screams, etc. That type of hysterical fit is not common in children, although

I shall have occasion later on to mention examples which might be referred to it. Much more commonly an hysterical fit in early life closely simulates epilepsy, and sometimes the simulation has been so close that it requires great care to distinguish one condition from the other. I can give you two remarkable examples :

Case 4.—Arnold H——, aged 14 years. About five weeks before he came under observation this boy fell down in the school playground in a 'fit,' from which he speedily recovered ; but since that time he had been subject to right-sided convulsions, which were getting more frequent. For the last two days he had not spoken.

He had had an attack of rheumatic fever at the age of ten, which had left him with a mitral lesion, but otherwise he had enjoyed good health. His parents were both said to be 'nervous.' There were four other healthy children.

He was a bright, healthy-looking boy, but exhibited complete 'mutism.' The fits recurred at intervals of a few minutes, and consisted in slight twitching of the right arm, leg, and face. The attack began with a slight grunt, and ended with a sigh. He was partially unconscious in the attack, but the conjunctival reflex was not entirely abolished. The eyes showed conjugate deviation in the attack, usually to the right, but sometimes to the left. The pupils were equal. He never bit his tongue nor passed water in a fit. There was slight weakness of the right arm, leg, and face ; and the knee-jerk on the right side was not so easily obtained as on the left. There was no anæsthesia, and the fundi were healthy. He readily obeyed commands, but wrote with great difficulty, and seemed to forget words and how to spell. He did not appear able to read print.

Under isolation with the use of faradism, suggestion, and cold shower-baths he quickly regained his speech, the fits stopped, and he left hospital quite well about three weeks after his admission.

Case 5.—William W——, aged 15 years, was seen in 1906 for 'fits,' which had begun three days previously. His mother had suffered from some sort of 'fits,' but the patient himself had always been healthy. The fits in his case had

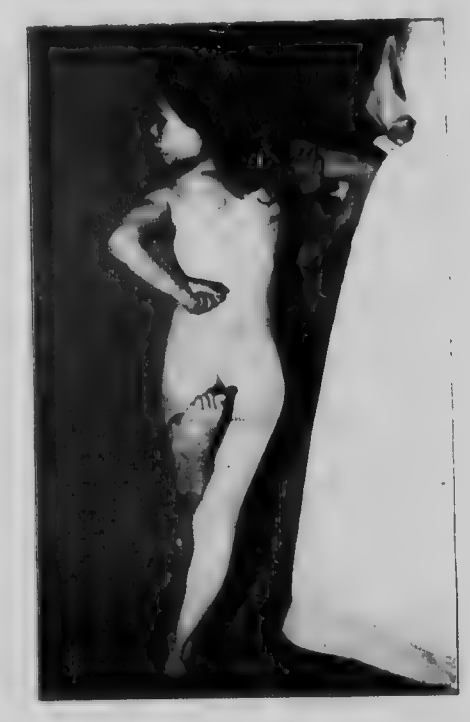

Fig. 30.
HYSTERICAL SPASM OF LEFT LEG.

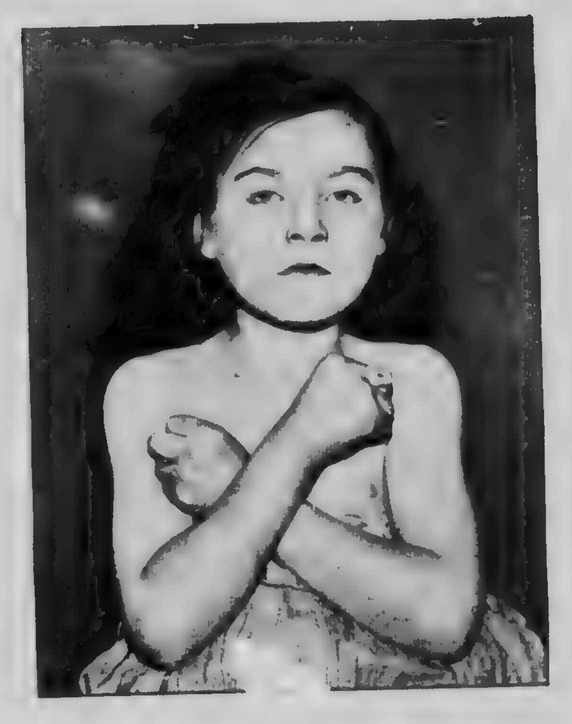

Fig. 31.
HYSTERICAL SPASM OF HANDS.

begun with 'stiffness of the jaw,' after which he was said to have lost consciousness. The fits had since been repeated nearly every quarter of an hour. He had never bitten his tongue in them, but once at least he had passed water during an attack. For the last twenty-four hours he 'had not spoken.'

On examination he seemed placid and intelligent, obeying commands readily, but quite mute. When a fit came on, the right side of the face twitched violently, and the hands were clenched, but showed no clonic spasm. There was no loss of consciousness, and the conjunctival reflex was not abolished. The pupils were dilated, the knee-jerks normal, and the fundi healthy. There was apparently no anæsthesia.

The fit lasted about a minute, and was repeated at short intervals.

He was admitted to hospital, and after about ten days recovered his speech, and the fits ceased.

In hysterical fits of this type I think mutism is a symptom of great diagnostic value. In both these cases its presence clinched the diagnosis.

Passing from fits, one comes to hysterical **contractures of joints.** These are rather common manifestations of hysteria in children, and I have seen several examples of them. They [illegible] follow injury of the joint, which has caused pain [illegible] to some degree of flexion, and that, owing to the hysterical basis, has become perpetuated and exaggerated, so that there is extreme spasm. One of the best examples I have seen was the following :

Case 6.—Jane K——, aged 11 years, was admitted to hospital for spasm of the hands of two to three days' duration. She had had several attacks of a similar sort before during the preceding year, and the feet also had been 'doubled up,' but the attacks had always passed off quickly in hospital. The patient herself said that the spasm began with a feeling of 'giddiness'; then the thumbs bent in, and the hands closed so that she could not open them. There was nothing noteworthy in the family history, and up till a year ago the patient had always enjoyed good health.

She was a well-developed girl, with a rather sullen expression. The fists were tightly closed, with the thumbs bent in and the arms bent across the chest. By gradual pressure the fingers could be unbent and straightened out, and then she could move them quite freely. The knee-jerks were exaggerated, but there was no ankle clonus. Sensation was normal and the viscera healthy. The visual fields were not tested.

The spasm quickly disappeared in hospital, but she had a typical attack of 'globus' whilst under observation.

It is important in cases of hysterical contracture not to overlook the fact that there may be a real organic basis for the condition. A child, for instance, may have commencing tubercular disease of a joint, or there may be some other disease of it, but, instead of responding with the ordinary stiffness and slight flexion, there is this exaggerated response, and you must beware in such a case of attributing the whole condition to hysteria.

The last motor manifestation I have to mention is **hysterical tremor.** There are various forms of such tremor. You may have a true intention tremor, which comes on only on attempts to use the affected part. That happened in the case of a boy, aged ten years, who had tremor of the hand as soon as he tried to write, although he used it for other purposes quite well. Sometimes the tremor consists in a series of clonic movements confined to one group of muscles—a repeated clonic spasm. There is little doubt that the condition called 'paramyoclonus' in children is hysterical, and I shall have occasion to describe to you immediately some cases affecting the respiratory system, which, I think, fall under this group. It is a characteristic feature of such hysterical spasms that they tend to stop during sleep.

SENSORY MANIFESTATIONS.

The next group of manifestations which one has to describe are the sensory ones. These, I think, are less common in children than the motor. There may be hemianæsthesia, as in the case of the girl I described (Case 2), or there may be anæsthesia of one limb of the sleeve type, as in the case of the boy with paralysis of the arm (Case 1). But in a considerable number of cases the hysteria is monosymptomatic, and there is paralysis of sensation without paralysis of motion. Hysterical hyperæsthesia is said to be commoner than hysterical anæsthesia in children, but I have not seen many clear examples of it, except those which are associated with contractures of joints, some of which are extremely painful. It is particularly difficult to be sure of the existence of an hysterical hyperæsthesia, and to exclude the possibility of underlying true disease, but not long ago I saw a case which I think may, perhaps, come under that heading. The patient was a little girl who was sent up with the history that not long before she had had an attack of whooping-cough, and ever since she had complained of pain in the abdomen, which almost prevented her walking. When she tried to walk she did so in a curious, stiff, cautious manner, and said it hurt her. She was examined thoroughly from every point of view—abdomen, spine, etc.—but we could not find any sign of organic disease, and it was noticed that after the examination was completed, and the child thought she was no longer under observation, she walked ever so much better. I think it is probable that this was a case of hysterical hyperæsthesia, but one should be very cautious in giving such an opinion unless one had had the case under observation for some time.

RESPIRATORY MANIFESTATIONS.

Passing from sensory manifestations, there is a large and interesting group of hysterical symptoms in children, which one may class as respiratory manifestations. Of course, mutism and aphonia are, in a sense, affections of the respiratory system, and both are common signs of hysteria in childhood. I have already described examples of mutism, and I would only point out here that it illustrates what I shall speak of more fully when I come to the question of diagnosis—namely, the tendency of hysterical manifestations to assume an exaggerated form. Instead of having a mere difficulty in speaking, as in aphasia, there is complete dumbness—that is to say, inability to speak at all—and it is this exaggeration of the disability which is so suggestive of hysteria. I would remind you also of another characteristic feature of cases of hysterical mutism and aphonia—namely, that the child is still able to cough, although he cannot talk. In true paralysis of the vocal cords that would be impossible. I shall have to refer again to this inconsistency of the symptoms in hysteria.

One sometimes meets with an **hysterical cough.** A good case of that kind was the following:

Case 7.—A boy, aged 15 years, was brought to me for a cough, which went on incessantly, wearing out the nerves of all the other people in the house. He had had a similar attack three years previously, which passed off in about a month. A year later he had another, lasting three weeks. Later on in that year he was hit on the head by a cricket-ball, and a week afterwards complained of pain behind the right ear. Nothing was found, but he kept holding a handkerchief to it all the time till the pain passed off, in about four weeks. Next year he got a worse attack of cough than ever, accompanied by great tachypnœa (120 per minute). The cough was attended by some frothy expectoration, but there were no signs in the chest or throat, and no rise of temperature. It is noteworthy that the cough ceased when he was asleep. This attack passed off entirely, and he returned to school, and remained well till the beginning of the present year.

He was a pale, rather poorly nourished boy, with a constant noisy cough, repeated every few seconds.

The lungs were perfectly healthy. The throat showed the remains of embedded tonsils, and the vocal cords were somewhat reddened. Examination with a probe showed some tender spots on the pharynx, which seemed to be the starting-points of the cough. The tonsils were enucleated, and the day after he did not cough at all. The following day he coughed as before, but next day he was much better. He subsequently made a good recovery.

Sometimes there is an **hysterical cry** or repeated utterance:

Case 8.—Constance B——, aged 7 years, was brought to the hospital in January, 1901, with the complaint that 'she made a curious noise,' and had done so, more or less, for the last five months, but it was getting more frequent, and occurred now nearly every day, lasting for from one to four hours at a time. The attacks were accompanied by pain in the throat and 'round the heart.'

She was the only child of a very emotional mother, precocious, and rather spoilt, but well developed and healthy looking. She had never had fits, but had sometimes had 'inability to walk,' and had fallen down on several occasions in the week before she came under observation.

During examination the cry came on; it took the form of a curious indrawing sort of crow, very difficult to describe, and was frequently repeated. Physical examination was entirely negative. She was admitted to hospital, but during her stay in the ward was perfectly normal.

The following was a case of a very similar sort:

Case 9.—A girl, aged 11 years, was seen in consultation in November, 1907. She was giving vent at short intervals to a sort of suppressed scream. This had been going on for six weeks, but it stopped during sleep and meals. She was very excitable and emotional, and easily burst into tears. The attack was said to have begun with pain in the tragus of one ear, passing round the helix. She was examined at the outset by an aurist, but nothing was found except slight redness of the drum, and the pain was supposed to be neuralgic, and various drugs were tried without success. She was undoubtedly a spoilt child, but I could find no signs of any organic disease, and her previous health was good.

She recovered quickly when removed to a Home.

Another very interesting respiratory manifestation is **hysterical dyspnœa,** or tachypnœa—rapid and laboured breathing without apparent cause. I have seen several such cases, of which the following are good examples:

Case 10.—Osmond F——, aged 8 years, was sent up to hospital with a history of laboured breathing for eight weeks following a series of 'fits,' which had been preceded by obstinate constipation. In the fits he was said to be unconscious, and threw his arms about, but did not pass water. He had always been delicate and constipated, and had a very poor appetite, living chiefly on biscuits.

On examination, he showed continuous panting respiration (thirty-six per minute), the inspirations being forced, but without involvement of the accessory muscles, nor was the diaphragm overactive. His breathing became worse when he was looked at. There were no signs of disease in the lungs nor elsewhere, but he exhibited some twitching of the nose and mouth. The knee-jerks were brisk and the fundi healthy, but there seemed to be some contraction of the visual fields. He was treated by faradism to the spine, and left hospital in a month much improved.

Case 11.—Edward J——, aged 15 years. Five days before he came under observation he got a blow on the head, which almost stunned him; since then his breathing had been difficult. He had always been nervous, but otherwise his health had been good. He breathed in a panting fashion, with forced inspirations at the rate of thirty-six per minute; the alæ nasi were active. There was no dyspnœa when he was speaking, and it was noticed that he could hold his breath for a long time then. The dyspnœa was worse when he was looked at, but disappeared during sleep. There were no signs of disease in the lungs or elsewhere, and no other stigmata of hysteria. He remained in hospital two weeks, being treated by isolation, suggestion, and cold shower-baths, with full ether narcosis on one occasion. He recovered sufficiently to be able to go to a convalescent home, but his subsequent history is unknown.

OCULAR MANIFESTATIONS.

Passing from these, one might next mention some ocular manifestations of hysteria. The first is *blepharospasm*, which is certainly hysterical in some cases. It usually begins with a slight conjunctivitis or ulceration of the cornea, which causes ordinary blepharospasm, and then the condition becomes exaggerated and perpetuated. I remember seeing a little girl, aged seven years, who exhibited this symptom in an extreme degree, one eye being screwed up as tightly as possible. She had had a good deal of conjunctivitis for some time previously, but it had now subsided, and under 'suggestion,' telling her she could open her eye and encouraging her to do so in the out-patient room, she opened it, and the spasm did not return.

You also meet sometimes with an hysterical *ptosis*. I have seen it associated with a *monocular diplopia*, a very curious condition. One also gets, amongst the ocular manifestations, *limitation of the field of vision*, but this is less constant in hysterical children than it is in adults.

VISCERAL AND MENTAL MANIFESTATIONS.

The last group of symptoms which one may mention are the visceral and mental manifestations of hysteria. These are much more difficult to define and to describe in the child, because one gets on the confines of congenital moral deficiency, or of mere bad temper, and also on the borderland of neurasthenia, and the precise distinction of hysteria from these conditions becomes difficult. Take the following case, for instance:

CASE 12.—Girl, aged 5 years. Since two years old has been subject to 'screaming fits,' which come on with the slightest provocation—*e.g.*, when asked to see a visitor or the doctor, or if the other children get something she wants. She throws herself down in the attack, and kicks and screams at the top of her voice. After the attack she is rather exhausted and dazed.

There was no family history of insanity, but the father was an extremely choleric and uncontrolled person. The patient had been late in walking and talking, but was said to be intellectually bright, although her attention easily flagged. Her doctor stated that she was physically sound, but I found it impossible to examine her, as she simply lay and screamed and kicked on the floor of my consulting-room.

A year later the attacks were said to be getting fewer.

It is very difficult to place such a case. Was it due to mere bad temper, to some moral defect, or to hysteria? In its want of control it certainly corresponds to hysteria, but I should not like to be sure about it. In other cases the child malingers or concocts groundless tales: or the condition takes the form of pure emotionalism, sometimes with a religious colouring, as in the following instance:

CASE 13.—Boy, aged 10 years. Had always been backward, and was obliged to go to a 'special' school, but went on all right till seven weeks before he came under observation, when for a week his 'arms and legs became rigid,' and he would not speak nor eat. This passed off, and was succeeded by fits of violence, in which 'he breaks and tears things' indiscriminately. Physically he seemed quite healthy and unusually well developed for his age, with a curiously deep voice; but mentally he was rather vacant and sullen. His father was of opinion that the services during Lent had upset him, as he always followed the Salvation Army, and would stand up at any time and preach or pray!

That is a case which in its history and symptoms certainly fits in with hysteria better than anything else. Sometimes hysterical children exhibit groundless fears or 'phobies,' but such cases are nearly allied to neurasthenia. I saw a little girl not long ago, for instance, who was panic-stricken at any sudden change in the weather. A thunder-storm particularly upset her, and every time one came on she would rush under the bed and hide herself. A rain-storm or a gale of wind had much the same effect. It is difficult to say whether or not such a case should be regarded as one of hysteria. Sometimes one meets with anorexia, and sometimes also with hysterical vomiting, but both these manifestations are rare in childhood.

Diagnosis.—The first step in the diagnosis is to *remember the possibility of the occurrence of hysteria in childhood.*

Many cases are overlooked because people do not realize that hysteria does occur in childhood or early life, and is not, as some are apt to suppose, a condition peculiar to the adult. Secondly, you have to remember that hysteria, not only in childhood, but in the adult, is always a thing *to be diagnosed with fear and trembling.* Mistakes are very apt to be made through overlooking the fact that there is some underlying organic condition present. You know that such a mistake is apt to be made in the case of young women suffering from early disseminated sclerosis, and it is also true of organic disease in children. Apropos of this, there is an interesting case recorded by Dr. Chaffey in the Reports of the Society for the Study of Disease in Children, vol. iii. It is that of a boy who was believed to have hysterical anorexia from emotional disturbance on the death of a favourite sister, but who finally proved to have a tumour of the brain.

Thirdly, remember that *symptoms and signs which cannot be voluntarily produced or simulated are probably not hysterical.* Most hysterical manifestations can be simulated or produced by an effort of the will if necessary. Take, for instance, the case of mutism. Anybody can be mute if he or she wants to, whereas such signs as changes in the optic discs, abolition of the deep reflexes, or true clonus, cannot be simulated or voluntarily produced, and as a matter of fact such signs are never hysterical, and if they are present you should not diagnose the case as one of hysteria.

Fourthly, *there is often something exaggerated or bizarre in hysterical manifestations*—what the French speak of as the 'massivity' of the symptoms—which is of help in diagnosis. An hysterical pain, for instance, is always an extreme pain, hysterical paralysis is always absolute paralysis, an affection of speech amounts to complete mutism, hysterical anæsthesia is profound anæsthesia, and so on. It is the profundity of the symptoms which is suggestive of hysteria. Fifthly, *it is suggestive of hysteria when the effect is out of proportion to the cause,* as when a child, for instance, gets a slight injury to a joint and develops a violent contracture. Sixthly, *inconsistency of the symptoms* with themselves is suspicious of hysteria. In the case of astasia-abasia, for instance, the child can kick its legs about in bed freely, although it cannot stand on them, as I have mentioned. It may be able to cough, but not to speak. Or the signs and symptoms may be inconsistent with anatomical facts. A paralysis, for example, has not an ordinary nerve or centre distribution at all, or an anæsthesia may not correspond to any distribution of peripheral nerves, or to central nerve areas, as, for example, in the sleeve type of anæsthesia. This is absolutely conclusive of hysteria. Seventhly, *a sudden onset or disappearance of the symptoms or causeless variations in their intensity* is suggestive of hysteria. The symptoms due to organic disease, as you know, usually come on gradually, and, as a rule, they do not go away suddenly. Finally, *the maintenance of a good state of the general health,* notwithstanding the severity of the local symptoms, is suggestive. The two boys with hysterical fits, whose cases I described to you (Cases 4 and 5), were examples of this. As soon as the fit passed off they were well, there was no elevation of temperature or interference with the general health, which would have been the case if they had had a succession of epileptic fits.

Prognosis.—With regard to prognosis, all I will say is that the prognosis of hysteria in childhood is good as regards immediate cure, certainly better than it is in the adult; but the difficulty is to prevent relapses, because of the patient's inborn constitution, and often because of his environment, and it may be part of your duty to change the surroundings, if you can, by sending the child away from home.

Treatment.—The first and most essential point in the treatment of hysteria in childhood is *isolation.* Get the patient into a home or hospital. Isolation is important, because it takes the child out of the unhealthy environment in which it has often been living, and that alone may be sufficient to effect a cure, as in some of the instances I have mentioned. Next, ignore, as far as you can, the condition of the child. Repeated examinations, demonstrations to students, and so forth, are bad for the patient. Wholesome neglect is the best attitude to adopt, and certainly the doctor and nurse should not be over-sympathetic, though, of course, the child should be treated with the glove of silk on the iron hand. In dealing with specific symptoms, what has been called the 'method of surprise' is good—that is to say, taking the patient unawares. In a case of astasia-abasia, for instance, put the child suddenly on its feet and tell it to walk. Sometimes under those circumstances the patient is taken aback and does walk, and then you have no trouble with it afterwards. But the objection to that method is the risk that if the child be not successfully taken by surprise you have not only failed to effect an immediate cure, but you have done something to perpetuate that symptom, and to make any subsequent treatment more difficult. But the manœuvre often comes off. 'Suggestion' is of great help—suggesting, for instance, that the child can move the limb, or that the pain has gone away, or that it can straighten out an affected joint, etc. With regard to other aids, I still believe in the old-fashioned treatment by cold shower-baths. Drugs, such as valerian, are perhaps helpful, and in some cases tonics may be necessary—iron, arsenic, and strychnine—so as to get the general health into good condition. It is extremely important, also, to see that the convalescence is made complete, and that the child is kept away from home for a considerable time until it is well out of the hysterical habit, as otherwise there is apt to be a relapse.

LECTURE XXI

THE PARALYSES OF CHILDHOOD

I. PSEUDO-PARALYSES.

GENTLEMEN.—When a child is brought to you who is alleged to be suffering from 'paralysis,' the first thing you have to determine is whether the loss of power is really due to some affection of the muscles or nerves, or whether it is merely a case, not of *inability* to use the limb, but of *disinclination* to do so because any attempt at movement causes pain. Cases of the latter sort are not very infrequent in early childhood, and they are spoken of as 'pseudo-paralysis' to distinguish them from genuine nervous or muscular lesions. There are three great causes of such false paralyses. Syphilitic epiphysitis is one, scurvy is another, and rheumatism is the third. I may say a word or two about each of these.

1. **Syphilitic Pseudo-Paralysis.**—Some of you may have seen a child in one of the cots lately who was brought to hospital because it had suddenly ceased to use the left arm. There was a vague history of injury, you may remember, and the case was at first regarded as being probably one of a lesion of the brachial plexus. On more careful examination, however, signs of congenital syphilis were detected, and I came to the conclusion that we were probably dealing with an epiphysitis of syphilitic origin. This conclusion was justified by the fact that under vigorous mercurial treatment the supposed paralysis quickly passed off. The case presented unusual difficulties, but, as a rule, you will readily make a correct diagnosis in such circumstances if you remember the existence of the condition. As a rule, syphilitic epiphysitis occurs in infants of only a

few weeks old—the oldest I have seen was six months—and it almost always affects several joints, particularly the joints of the arms; indeed, I do not remember to have seen a syphilitic pseudo-paralysis of the lower limb. The proper treatment is to place the limb in a splint, and to push anti-syphilitic treatment. If this plan is adopted, a speedy cure almost always results.

2. **Scorbutic Pseudo-Paralysis.**—The other day a child ten months old was brought to the receiving-room for loss of power in one leg, and was sent down to out-patients' with the diagnosis 'Paralysis. ? cause.' On examining the infant, one was at once struck by the great tenderness of the limbs, and on inspecting the gums they were found to show slight sponginess round the incisor teeth, whilst inquiry into the history revealed the fact that the child had been fed on condensed milk and a patent food. Anti-scorbutic treatment was adopted, and when seen a week later the child was using the limb freely.

Here, again, the apparent paralysis was really a mere disinclination to use the limb, from pain. In contrast to syphilitic pseudo-paralysis, it is the lower limbs which are most apt to be affected in this condition. The extreme tenderness of the limb when handled should usually put you on the right track, and often you will find also some degree of thickening round the lower end of the femur. Your diagnosis will be confirmed by finding sponginess of the gums and a history of the child's having been fed on the sort of diet which is apt to produce scurvy. The diagnosis once made, the treatment is obvious, and its results usually brilliant.

3. **Rheumatic Affections,** particularly of the lower limbs, may also sometimes simulate paralysis. I have known this happen, especially when the tendo Achillis, or hamstrings were involved. Here it is older children who are affected, never infants, and the immobility is rarely complete, but is chiefly manifested by an inability to walk properly. You will usually find some signs pointing to rheumatism elsewhere, or a history of its occurrence in the past, and the use of salicylates and local friction with a liniment speedily bring about a cure.

Finally, it is always well to bear in mind the possibility that paralysis may be simulated by a purely **surgical affection.** I have known a distinguished physician, for instance, be completely nonplussed by a case of double congenital dislocation of the hip, and ordinary simple epiphysitis or hip-disease or dislocations may easily lead you into error.

II. TRUE PARALYSES.

Assuming that you are satisfied that you have to deal with a case of true and not of simulated paralysis, the next thing you have to determine is the **seat of the lesion.** In this diagram (Dia. 9) there is shown the course of a motor fibre from its starting-point in the cerebral cortex to its termination in a muscle, and there is indicated also the different lesions of which it may be the subject at various points in its course. The first step to determine is whether the lesion affects the upper or lower motor segment. This is not difficult to do if you remember that upper segment lesions are characterized by spasticity, with exaggeration of the deep reflexes and absence of muscular wasting, whereas lower segment lesions exhibit flaccidity, with diminution of the deep reflexes and the presence of wasting. We may therefore consider first the lesions of the upper segment, known as—

THE SPASTIC OR CEREBRAL PALSIES.

According to their period of onset, these may be divided into three groups :

1. Pre-natal—*i.e.*, occurring *in utero.*
2. Natal—*i.e.*, occurring at the moment of birth.
3. Post-natal—*i.e.*, occurring some time after birth.

Pre-natal cases are usually the result of malformation of part of the brain—a condition to which the term *porencephaly* is applied—or of an arrested development of the cortical cells (*agenesis corticalis*).

The **natal cases** are due to an injury to the brain during parturition. The nature of the injury is often obscure, but one may safely regard it, in many cases at least, as

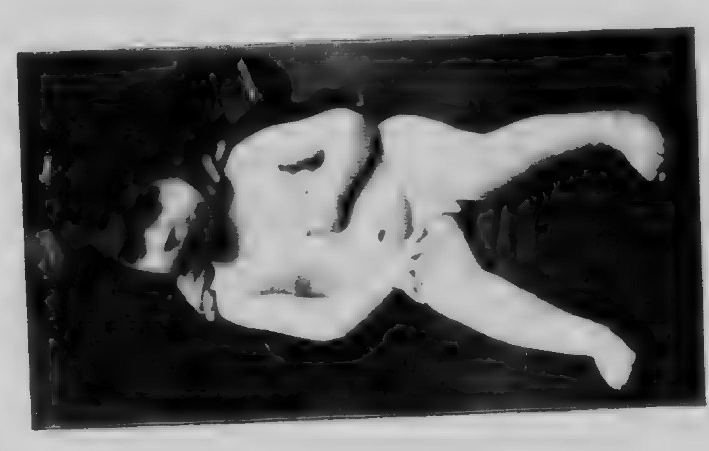

Fig. 34.

LEFT HEMIPLEGIA, DATING FROM BIRTH.

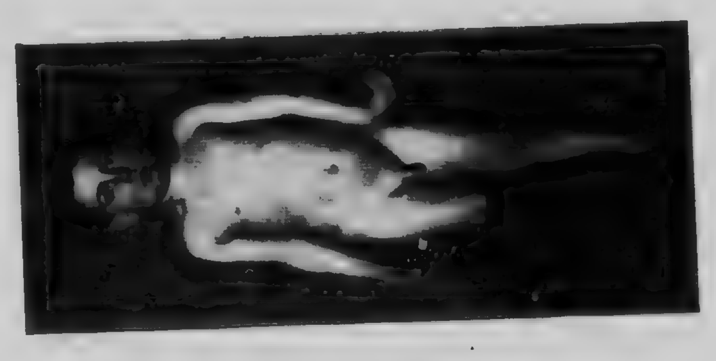

Fig. 33

LEFT HEMIPLEGIA, SHOWING MARKED SPASM OF ARM.

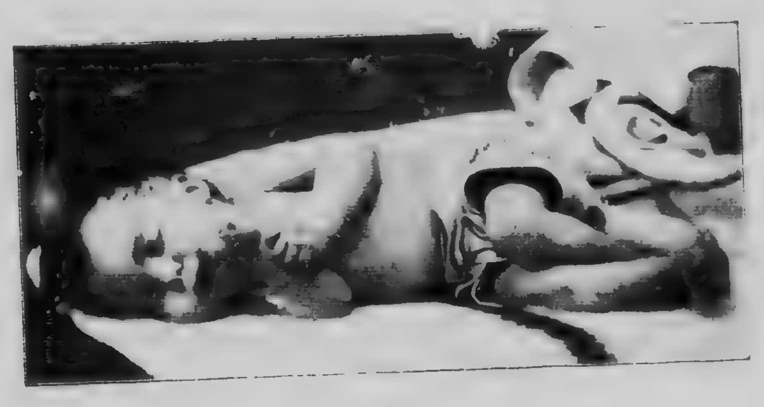

Fig. 32.

CASE OF DIPLEGIA, SHOWING GENERALIZED RIGIDITY AND INTRA-UTERINE POSITION.

being due to a meningeal hæmorrhage. Very commonly one finds that such children have been born prematurely, and only resuscitated with great difficulty. Instrumental delivery is by no means always a factor.

Cases which come on some time after birth* (**post-natal**) are usually hemiplegic, and may be due to the same causes as produce hemiplegia in adults—*e.g.*, embolism, syphilitic thrombosis, or even hæmorrhage. Sometimes, at least, they are the result of surface lesions—*e.g.*, venous thrombosis—and they may also be due to a polio-encephalitis affecting the motor area analogous to the poliomyelitis which is so common in the gray matter of the cord.

As regards its **distribution,** the paralysis may be :

(1) Hemiplegic (involving one arm and leg) ;
(2) Paraplegic (involving both legs) ;
(3) Diplegic (involving all the limbs) ;
(4) Monoplegic (involving one limb only) ;

in that order of frequency, and depending upon the site and extent of the lesion.

The paraplegic cases are sometimes spoken of by the special name of Little's disease—a term which should appeal to all of you here ; for Dr. Little was at one time physician to the London Hospital, as well as founder of the Royal Orthopædic Hospital.†

I have used the term **Diplegia** to signify a paralysis affecting all the limbs. You will often find it employed, however, to signify any bilateral affection of the cortex which results in paralysis, and if used in that sense it would cover paraplegia as well. There are several varieties of diplegia. The typical form in which all the limbs are affected is also spoken of as *generalized rigidity*, and a good example of it is shown in Fig. 32. Such cases are usually of pre-natal origin. At other times, again, it results from a progressive degeneration of the cortical cells coming on some time after birth—a condition sometimes described as one of 'premature senility' of the neurons. Such cases often occur in various members of the same family. In yet another group the progress and symptoms closely resemble those of G. P. I., and in that case the disease usually starts during adolescence, and is probably of syphilitic origin. To the diplegias also belong the cases of

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* It is well to remember that the history is not always a safe guide as to whether a case is of natal or post-natal origin, for there can be little doubt that in many cases which really date from birth the paralysis is not noticed till the child tries to walk.

† Little's original paper was published in the Trans. of the Obstetrical Soc., 1861, vol. iii., p. 297.

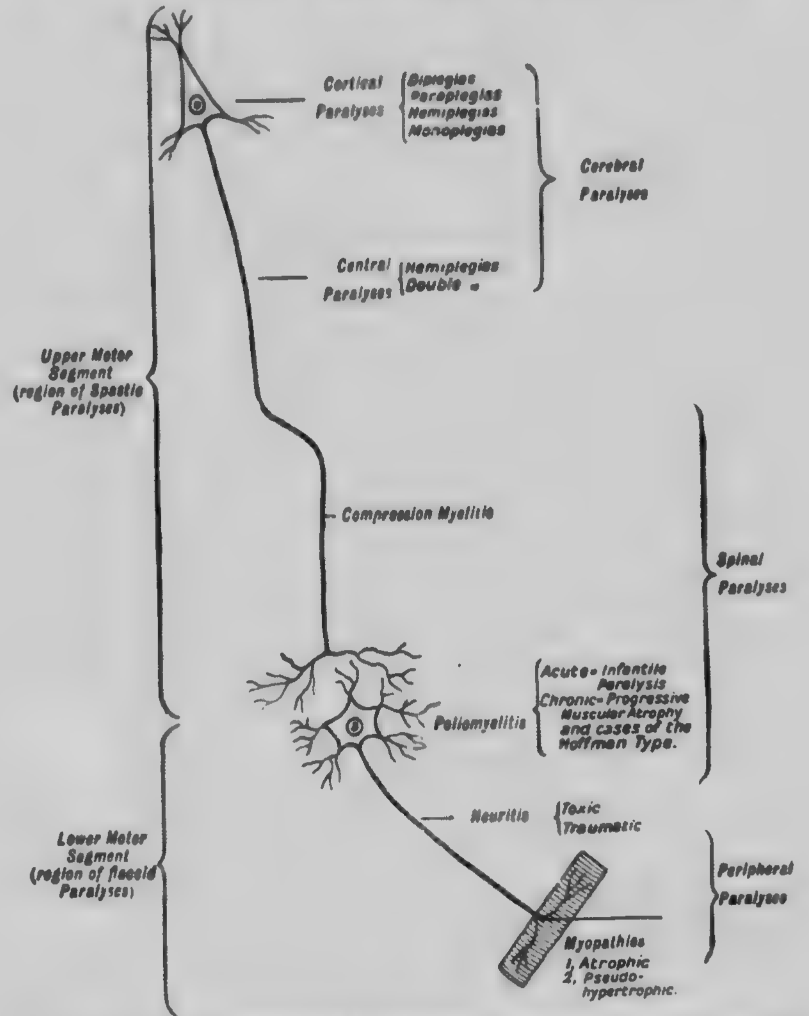

DIAGRAM 9. -TO SHOW THE SITE OF THE LESION IN THE PARALYSES OF CHILDHOOD.

amblyopia with mental deficiency sometimes met with in Jewish families, to which I shall have occasion to refer in a later lecture.

You might suppose that it would be impossible to distinguish a case of diplegia from one of double hemiplegia, but it is not so. In diplegia there is always more rigidity than paralysis, in hemiplegia the reverse. In hemiplegia, again, the arm is always more affected than the leg ; in diplegia this is not observed. As regards the relationship between the distribution of the spastic palsies and the time of their causation, it may be said that the hemiplegias are usually of post-natal development, the paraplegias of natal production, whilst the diplegias may be of either pre-natal or post-natal appearance. Monoplegias are so rare that I cannot tell you what is their usual time of causation.

COMPLICATIONS OF THE SPASTIC PALSIES.

You will find, gentlemen, that the spastic palsies not merely produce a greater or less degree of impairment of motor power ; they also bring in their train certain remoter consequences. Two of these are specially prominent, and should be remembered when you are giving a prognosis. One is mental impairment, the other is fits.

1. **Mental Impairment.**—You will not be surprised to find that any severe and extensive cerebral lesion in a young child is apt to be followed by some impairment of the mental functions, and, in accordance also with what one would expect, one finds that the earlier the lesion makes its appearance, the more apt is such impairment to be manifested. So far as I have been able to observe, diplegics are invariably imbecile ; they become, indeed, practically dements, whilst paraplegics are more apt to suffer than hemiplegics. The degree of mental disturbance may vary from a mere slight degree of 'eccentricity' to a condition of absolute imbecility.

2. **Fits.**—If you inquire into the history of cases of spastic paralysis of post-natal appearance, you will often be told that the paralysis set in in consequence of a fit. Whether the fit in such a case is really the cause of the

paralysis, or whether both fit and paralysis are the common result of a cerebral lesion, I am not prepared to say; but of this there can be no doubt—that in many cases the fits tend to recur in such children, or to make their appearance sooner or later, even although they were not present from the outset. The fits in such cases are typically epileptic in type, and often occur with great frequency and severity, and are of special importance, because there can be no doubt that they tend still further to impair mental development. They are probably more apt to occur in those cases which are the result of a surface lesion, especially when it happens to be both extensive and of early production.

POTT'S PARAPLEGIA.

Cases of spastic paraplegia are sometimes brought to one with the history that the paralysis has come on gradually. In such cases it is always advisable to examine the back very carefully for signs of spinal caries, particularly in the cervical and upper dorsal regions, for **compression myelitis,** as it is called, is not an uncommon sequence of spinal caries in these regions. Here, again, it is the upper motor segment which is involved—*i.e.*, the motor fibres on their way to the anterior cornual cells of the lower dorsal and lumbar parts of the cord—and that being so, the paralysis is, of course, spastic in type. It differs, however, in several respects from the spastic paraplegia of cerebral origin which I have already described. In the first place, the history usually makes it quite plain that the symptoms have developed *slowly*, whereas, as we have seen, in the so-called Little's disease the paralysis has almost always dated from birth. In the second place, the sphincters are usually involved early in Pott's paraplegia; indeed, overdistension of the bladder, with consequent cystitis, is one of the unpleasant results which, unless the doctor is watchful, the affection of the sphincters may bring about. The paralysis is sometimes preceded by pains referred usually to the chest or abdomen, and you will find it a good rule in every case in which a child complains of persistent abdominal pain to examine the back.

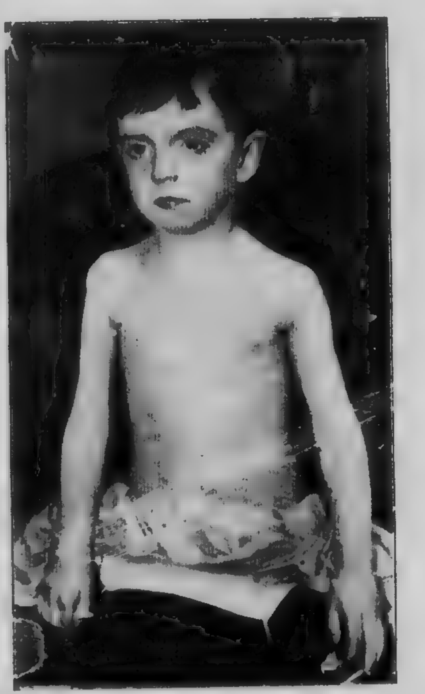

FIG. 35.

INFANTILE PARALYSIS, AFFECTING RIGHT DELTOID.

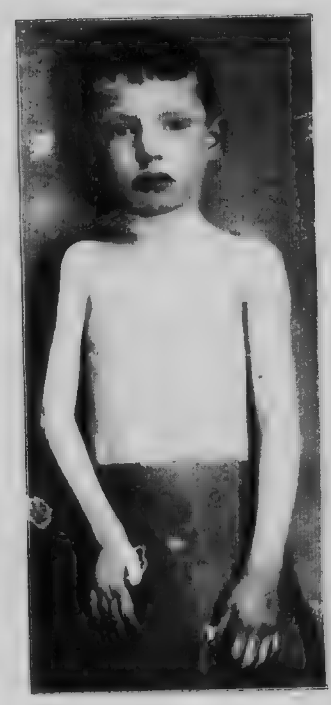

FIG. 36.

INFANTILE PARALYSIS, RESULTING IN SHRINKING OF RIGHT ARM.

You should not take too gloomy a view of these cases. It is surprising how completely the paralysis may be recovered from if the child is kept for a sufficient time at perfect rest. Rest, indeed—absolute and prolonged rest—is the keynote of their successful management.

LESIONS OF THE LOWER MOTOR SEGMENT.

The most typical form of paralysis which results from a lesion of the lower motor segment is **infantile paralysis,** which is, as you know, the consequence of anterior poliomyelitis. Now, the first remark I have to make about 'infantile' paralysis is that it is a rather badly-named disease.

It is not, indeed, a characteristic disease of *infancy* at all, for it is but rarely met with before the end of the first year. The great majority of cases occur in the second and third years. You will usually have but little difficulty in recognizing a case when it is brought to you. The flaccidity of the limb, the loss of the deep reflexes, the wasting of the muscles, and the presence of the reaction of degeneration, all proclaim it at once as due to a lesion of the lower motor segment, and the only thing for which you can mistake it is a paralysis due to interference with a peripheral nerve. How such an error is to be avoided I shall point out shortly, when I come to speak of the peripheral palsies of childhood.

Although a fully-developed case is not difficult to recognize, you will often be puzzled should you happen to see the patient during the active stage of the disease. It will be but rarely, however, that you will have that opportunity. The onset of anterior poliomyelitis is often marked by but little in the way of general disturbance of health. A little fever, perhaps a disinclination for food, and a 'going off his legs,' may be the only indications of the serious mischief which is on foot. Sometimes, however, the disease sets in with more of a fracas. I remember being asked to see a boy of five by one of my colleagues here, who had been admitted to the hospital suffering from severe fits. When I examined him he was almost in a *status epilepticus*, passing from one severe general convulsion to another, with brief intervals of only partial consciousness. A careful examination revealed no signs of disease, and one was driven to make the tentative diagnosis of convulsions probably of reflex origin from the bowel. In a day or two, however, the mystery was cleared up, for the boy emerged from his fits, but with complete paralysis of the shoulder group of muscles on one side. It had been a case of anterior poliomyelitis ushered in by convulsions.

Another mode of onset which may puzzle and mislead you is that in which the initial stage of the paralysis is attended by great **hyperæsthesia** of the affected limb. I remember being quite deceived by the first case of this sort which I saw. It was that of a little girl who was under my care whilst I was in temporary charge of one of the wards at Great Ormond Street. Her left leg seemed to be paralyzed, but whenever one attempted to examine it the child seemed in such pain, and screamed so loudly, that one was obliged to desist. Not being at that time aware that infantile paralysis might be accompanied by great tenderness in its early stages, I was inclined to think that one had to deal with one of those pseudo-paralyses which were described at the beginning of this lecture, and discussed the possibility of mischief in the neighbourhood of the hip-joint. Examination under an anæsthetic, however, revealed nothing, and by-and-by the tenderness passed off, and the child was left with an ordinary infantile paralysis of some of the muscles of the thigh.

As regards the **prognosis in infantile paralysis,** one can always safely say that, after the acute stage has passed off, any change which takes place will be in the direction of improvement. The extent to which the affected muscles will be finally disabled can be gauged better by the electrical reactions than by any other test. The more completely and extensively faradic excitability is lost, the more irreparable is the damage which has been done.

I shall discuss the **treatment** of infantile paralysis and the deformities which result from it along with that of paralyses in general.

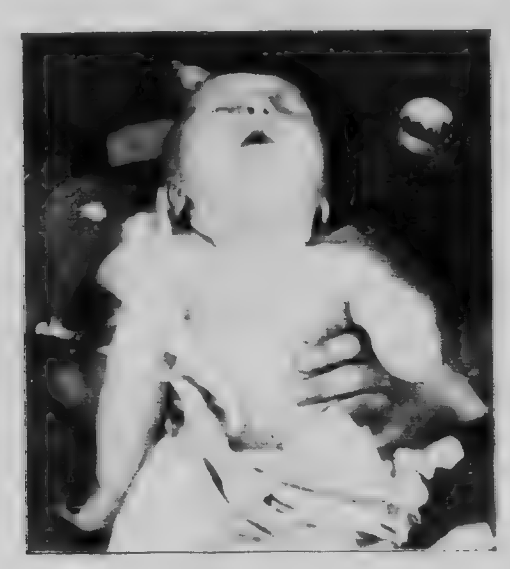

Fig. 37.

ERB'S PALSY.

Note characteristic position of right arm.

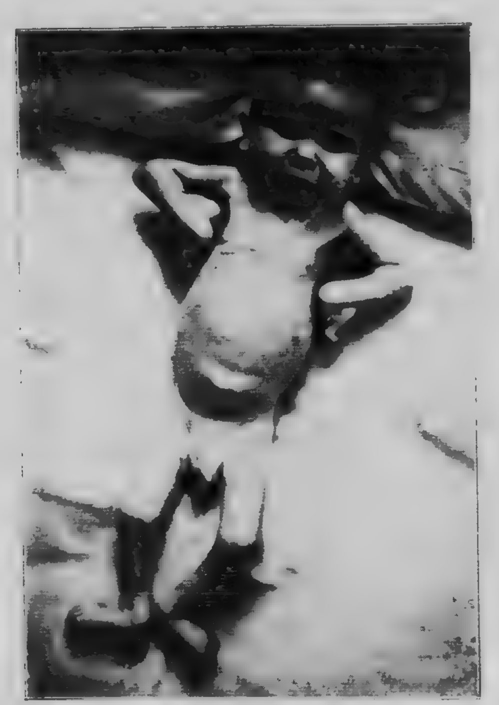

Fig. 38.

HOFMANN TYPE OF PROGRESSIVE MUSCULAR ATROPHY, SHOWING GREAT WASTING OF ALL MUSCLES, ESPECIALLY OF THE INTERCOSTALS.

CHRONIC ANTERIOR POLIOMYELITIS.

Chronic anterior poliomyelitis leading to progressive muscular atrophy is a rare disease in childhood. Hofmann has described* a special type of it which occurs in families, and of which the case depicted in Fig. 38 was an example. This child, a boy four months old, was brought to see me at Great Ormond Street with the history that he had become gradually paralyzed within the last three months. The mother stated that an exactly similar fate had befallen two other of her children at the same age. On examination the infant was found to exhibit complete flaccid paralysis of all the muscles except the diaphragm, so that he lay helpless on his back, breathing with difficulty and occasionally giving vent to a feeble cry. The atrophy of all the muscles is well shown in the photograph, and a few fibrillary tremors could be observed. The child died outside shortly afterwards, but, unfortunately, no examination could be obtained. Such cases are exceedingly rare, and belong rather to the curiosities of pediatrics, for you may spend a long life in busy practice and never meet one of them.

PERIPHERAL PALSIES.

Paralysis from lesions of the peripheral nerves are not very uncommon in childhood, and may be divided into the two great groups of (1) traumatic and (2) toxic. The best examples of the former are the 'obstetrical palsies,' which result from injury to nerves at birth; the latter are typified by post-diphtheritic paralysis. It will be well to consider each group separately.

1. **Obstetrical Palsies.**—These may affect the arm, the leg, or the face. Those of the arm are the commonest and best known, and usually take the form of **Erb's paralysis,** which is the result of an injury of the fifth, sixth, and seventh cervical nerves at the root of the neck. These nerves supply, as you will remember, the deltoid, biceps, brachialis anticus, and supinator longus, as well as the

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* *Münch. Med. Woch.*, 1900, xlvii., p. 1649; and *Deut. Zeitsch. f. Nervenheilk.*, 1893, iii., p. 427.

external rotators of the shoulder (rhomboids, supra- and infra-spinatus, and teres minor). When these muscles are paralyzed, their opponents have it all their own way, with the consequence that the arm assumes a very characteristic position, being held limp by the side, fully extended and pronated, and rotated inwards at the shoulder, so that the palm of the hand looks backwards and a little outwards—much in the position, as it has been remarked, in which a man places his hand when expecting a tip. (The characteristic position is well shown in Fig. 37).

The injury to the nerves in these cases is probably the result of pressure or of laceration, and the labour will almost always be found to have been a difficult one. This may be the reason why the condition is commoner in boys. Of fourteen consecutive cases of which I have notes, nine were males and five females. It is commonly said that most cases are the result of a breech presentation, the injury being inflicted in the delivery of the after-coming head, but in only four of my cases was the child born by the breech. Nor can one attribute it to injury by forceps, for in ten of my cases the child was born without the aid of instruments. Of my own cases, eight were right-sided, four left-sided, and two were bilateral; but other observers state that the preponderance of cases is on the left side.

In a typical case the condition can be recognized at a glance, but as all the above-mentioned muscles are not always equally affected, many atypical forms are met with. These you may have difficulty in diagnosing from infantile paralysis, and, in the absence of a history, I do not know of any way in which you could be sure which it was you were dealing with. Usually, however, you will be clearly told that the paralysis was noticed at or shortly after birth, and that should keep you right. The future of such cases is difficult to forecast. The majority probably recover to a very great extent, for typical examples of the paralysis are very rarely met with in adult life; but often, no doubt, some degree of weakness of the shoulder muscles remains. Your estimate of the chances of recovery in any given case must be based on the same data as were laid down for your guidance in the prognosis of infantile paralysis.

Obstetric paralysis affecting the **leg** is said to occur, but I have never seen an example of it. **Facial paralysis** from this cause, on the other hand, is not very uncommon, and is often the result of the pressure of the blades of the forceps. Usually it passes off very quickly, but Edgeworth has recorded* one bilateral case which was still very marked at the age of seven.

The treatment of these affections I shall come to later.

2. **Toxic Neuritis.**—With the exception of the post-diphtheritic form, toxic neuritis is very uncommon in childhood. The forms which you are familiar with in adults as due to lead, alcohol, and arsenic you will hardly ever see in the child.

Occasionally one sees cases of **multiple neuritis** in children, the cause of which is difficult to ascertain. Some of these are vaguely spoken of as being 'rheumatic.' They are not difficult to recognize, for, besides presenting all the ordinary characters of a lower segment lesion (wasting, loss of deep reflexes, reaction of degeneration), they are characterized by being widespread and usually symmetrical tions, which at once distinguishes them from cord lesions. her—at the outset, at least—they present more or less impairment of the sensory functions, as well as of the motor.

Post-diphtheritic paralysis, which is by far the most important of the toxic neuritis group in childhood, I do not propose to speak of very fully, for its study is always taken up along with that of diphtheria. Its chief peculiarity, you remember, is its tendency to affect certain nerves more than others, paralysis of the soft palate being one of its earliest and most constant manifestations. Paralysis of the muscles of the eye is also not uncommon, nor, unfortunately, is that of the respiratory muscles and heart, for when these are affected the patient's condition is often very dangerous.

The cases of post-diphtheritic paralysis which you are most apt to overlook are those in which the child is brought to you for weakness of the legs, with, perhaps, inability to walk properly. In such a case there may be no clear history of diphtheria obtainable and no evidence of paralysis of the soft palate, and I have known great difficulties in diagnosis arise. The absence of the knee-jerks, however, ought to keep you right, for though this might be due to infantile paralysis, yet the latter is not so likely to be symmetrical, and the wasting is usually much more pronounced. It is well to remember, too, that post-diphtheritic paralysis is rare before the age of two, whilst the majority of cases of infantile paralysis occur in the second year.

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* *Brit. Med. Journ.*, 1894, i., p. 11.

FACIAL PARALYSIS IN CHILDHOOD.

The ordinary Bell's palsy so frequently met with in adults, and usually attributed to the effects of cold, is not at all common in childhood. When a child is brought to you with facial paralysis, you should think of two things: (1) That it may be an obstetrical palsy; (2) that it may be the result of middle-ear disease.

Of the first of these I have already spoken, and have pointed out that the paralysis in such cases always dates from birth, and is usually transitory, and you should have no difficulty in diagnosing it.

Facial paralysis which results from **disease in the middle ear** is by no means uncommon, and may sometimes even be bilateral; but its recognition is easy, for there is always a history of a discharge from the ear on the affected side. When both nerves are affected, the characteristic appearance of the face known as 'tapir-mouth' is produced.

Clearing out the mastoid in these cases often gives brilliant results, and I have known the paralysis begin to pass off within a few hours of the operation. Sometimes however, a greater or less degree of permanent interference with the functions of the nerve results.

MYOPATHIES.

Peripheral palsies the result of affections of the muscles themselves are almost peculiar to childhood, and may be conveniently spoken of as 'myopathies.' Whether the disease of the muscle in these cases is truly primary, or, as

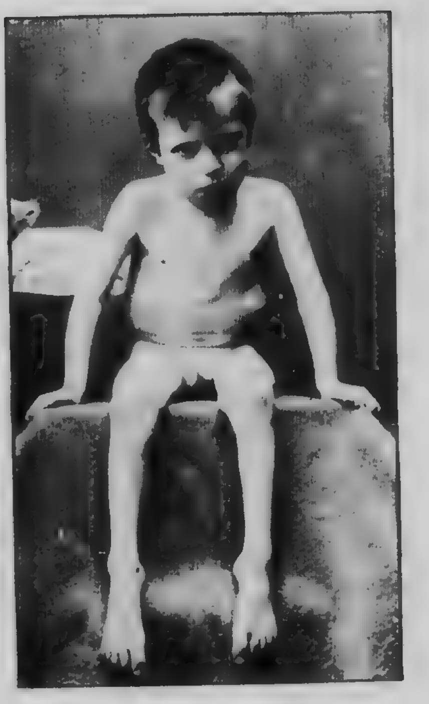

FIG. 39.

PERIPHERAL NEURITIS, PRODUCING 'FOOT-DROP.'

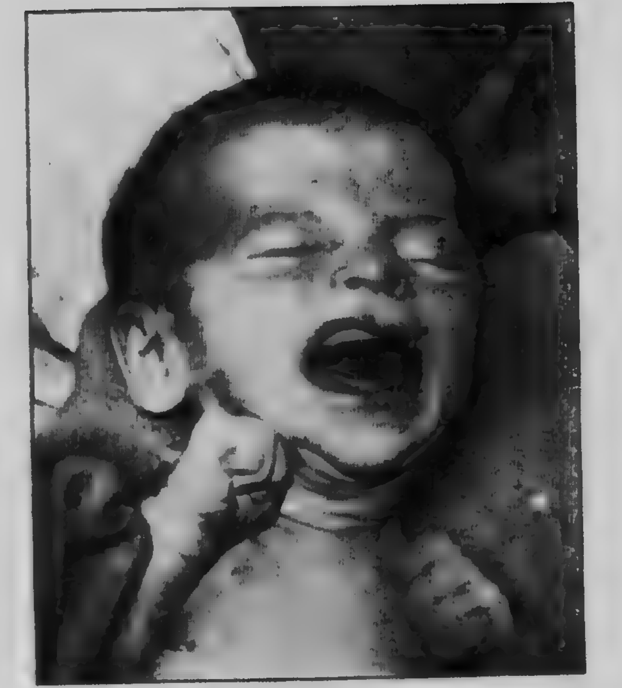

FIG. 40.

OBSTETRIC FACIAL PALSY.

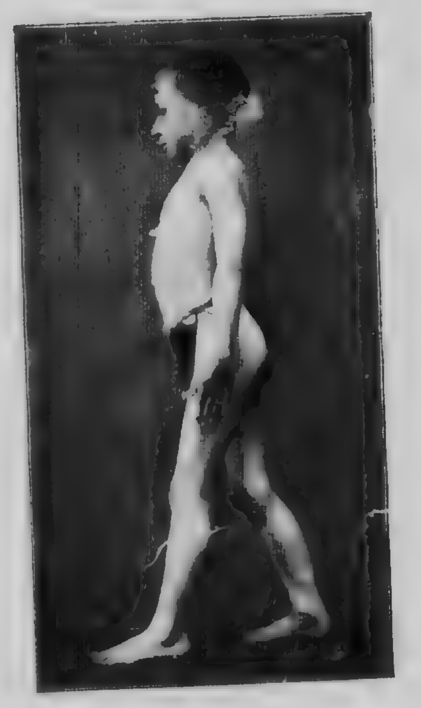

FIG. 41.

EARLY CASE OF PSEUDO-HYPERTROPHIC PARALYSIS, SHOWING ENLARGEMENT OF CALVES AND SLIGHT LORDOSIS

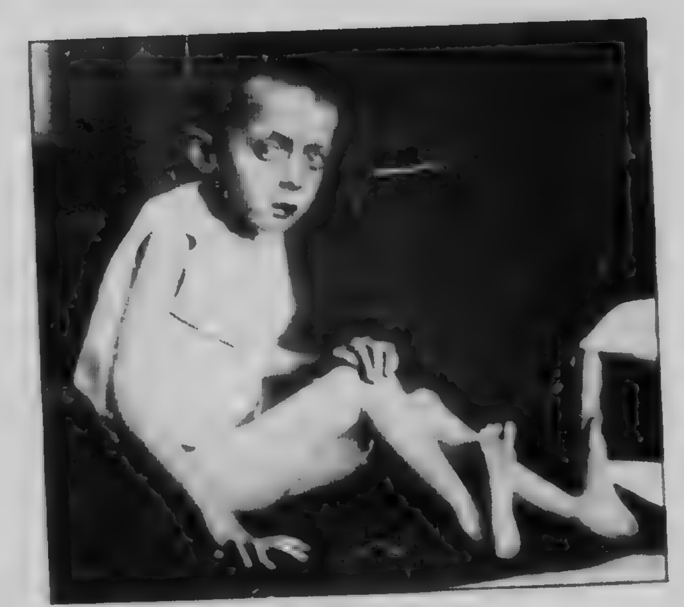

FIG. 42.

MYOPATHY OF ATROPHIC TYPE.

most neurologists are now beginning to think, is really the result of an affection of the nerve cells of the cord, is a recondite pathological question into which we need not enter. In any case, the paralyses of this group are far from common, even in childhood, and I only mention them here for the sake of completeness.

Cases of myopathy may be roughly divided into two groups—(1) Those in which the affected muscles, or some of them, are apparently enlarged and thickened ; (2) those in which marked muscular atrophy is present.

1. **Pseudo-hypertrophic paralysis** is the classical example of the first group, and its peculiarities are so carefully dealt with in every text-book that I need not spend further time upon it.

2. The **atrophic cases** are divided, often rather arbitrarily, into different groups, according to the muscles affected. Thus, one speaks of the 'facio-scapulo-humeral' type, of the 'peroneal' type, and so on. In some of these cases the atrophy of the muscles and loss of power have been noted at or very soon after birth (infantile type), in others they had come on gradually at some period of childhood. The degree of atrophy varies, being in some cases extreme, and various deformities of position may result, whilst the hands and feet sometimes look curiously long and thin (Fig. 42). The electrical reactions may be diminished quantitatively, but usually show no qualitative alteration which marks the condition off from anterior poliomyelitis and neuritis, whilst the absence of fibrillary contraction distinguishes it from progressive muscular atrophy. Little if anything, unfortunately, can be done to improve them, but they often exhibit but slight tendency to progress, and may survive in fair health for many years.

Apparent paralysis, the result of congenital absence of muscles or bones, may be mentioned, but if you remember the fact of its existence you are not likely to be deceived by it.

TREATMENT OF THE PARALYSES OF CHILDHOOD.

I can promise you, I am afraid, but little satisfaction in your treatment of the paralyses which have been described in this lecture. For the most part, they depend upon organic lesions of the nervous system which it is quite beyond your power to modify or remove, and at best you can only hope in most cases to mitigate their effects.

In the treatment of the **spastic paralyses** you will find a great ally in the orthopædic surgeon, who can do much by instruments and supports of various sorts and by operation to increase the usefulness of the affected limbs. The detailed consideration of such procedures, however, would be quite beyond my province. For the fits which often ensue in these cases bromides and chloral may be given, and although I have seen it stated that bromides do no good in these cases of organic lesion of the brain, yet I can only say that I have occasionally seen great benefit from their continued use. Further, by diminishing the number and severity of the fits, one does something at least towards arresting the supervention of mental impairment, for there can be no doubt that frequent and severe fits seriously impair the intellectual functions. Should mental deficiency be already present, it must be treated on the lines which I shall lay down in a lecture on mental deficiency in general.

For **Pott's paraplegia** I have already said that the chief treatment is rest, along with such support by splints or otherwise as you are accustomed to see used in spinal caries. Iodide of potassium in *large* doses is also strongly recommended by some writers in these cases. It perhaps acts by aiding in the absorption of the inflammatory material which is compressing the cord. Attention to the bladder is always imperative. I have known serious cystitis set up by a neglect of this important matter.

Infantile paralysis is now known to be an infectious disease, and therefore in the acute stage the patient must be strictly isolated and a disinfectant spray used for the throat and nose, which are the probable channels of infection. Pain may be relieved by opium or aspirin, and urotropin or helmitol should be administered, as these drugs pass quickly into the cerebro-spinal fluid, and may help to destroy the infective agent. The limb should be placed on a light splint, with the affected muscles in a position of relaxation. When the acute phase is over, massage and electricity are useful. The value of the latter, however, must not be overrated. It can certainly do no good in the long-established cases in which the paralysis has existed for some months. It has also the disadvantage of being very apt to frighten the child.

The nutrition of the affected limb can be helped by warm covering and by douching with salt and water, followed by vigorous friction with a rough towel. Any degree of voluntary movement is far more valuable than any amount of massage, and the child should therefore be encouraged to use the limb as much as possible. This can be done by putting a little collar of bells round the wrist or ankle, and getting him to ring them by moving the arm or leg; the finer movements can be developed by teaching him to prick out patterns with a pin, or by any similar device which the intelligence of the mother can suggest.

For the club-foot and other deformities which so commonly result in infantile paralysis, you must again have recourse to surgical aid.

For **neuritis** in its early stage rest and symptomatic treatment must be your rule. Later on, electricity, massage, and strychnine will be of help. In the case of the special injury of nerves which causes **Erb's palsy** it has been recommended that if the lesion be recognized at birth the arm should be put up for a month in the same position as for fractured clavicle, in the hope that this may help the repair of the nerve-trunks. Later on it must be treated like any other neuritis. Some successful cases, in which the nerve-trunks were sutured in this form of paralysis, have been recorded,* and perhaps this method of treatment may become more widely used in the future.

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* See Kennedy, *Brit. Med. Journ.*, February 7, 1903.

LECTURE XXII

MENINGITIS

GENTLEMEN,—Meningitis is a disease which is comparatively common in childhood—much commoner, certainly, than it is in the adult—but it is one which is peculiarly difficult to describe in a lecture, and I feel far from confident of my ability to give you anything like a clear description of it. This is due to the great variability in the symptoms which the disease presents—a variability which is to be ascribed to variations in the extent to which the meninges are involved in any given case, and to the variety of the agents which may excite the inflammation. I feel pretty confident that in the future, when our knowledge is more extensive and exact, we shall come to a bacteriological classification of the disease, though at present I have thought it advisable to abide by the recognized divisions. You must remember, however, that we already know that several different micro-organisms are capable of exciting inflammation of the membranes of the brain and cord. Chief of these are the *Diplococcus intracellularis meningitidis* of Weichselbaum (shortly known as the meningococcus), the tubercle bacillus, the pneumococcus, and the pyogenic organisms, and any of these may be found present alone, or 'mixed infections' may occur.

GENERAL SYMPTOMS.

No matter what its exciting cause may be, inflammation of the membranes of the brain and cord results in the production of a group of **symptoms which are common to all forms of meningitis,** though one or more of these symptoms may dominate the picture in any given case of the disease. The symptoms may be roughly divided into two groups : (1) Those which result from irritation of the brain ; (2) those which are the consequence of increased intracranial pressure.

1. Amongst the **irritative symptoms** the most prominent are mental irritability— anifested in its slighter degrees by mere peevishness, and in its more pronounced forms by signs of great irritability when roused—screaming, headache, convulsions, vomiting, irregularity of the pulse and respiration, photophobia and contraction of the pupils, and sometimes optic neuritis. There is also apt to be an increase of the 'tone' of the muscles, showing itself by exaggeration of the reflexes, by muscular twitchings, by the presence of Kernig's sign and, in extreme cases, by opisthotonos.

2. Of the '**pressure signs**' the most important are lethargy and drowsiness culminating in coma, bulging of the fontanelle, slowing of the pulse and respiration, and dilatation of the pupils.

The special symptom known as '**head retraction**' is often one of the most prominent features in meningitis, and it is difficult to know to which of the above groups to refer it. The photograph (Fig. 43) will give you a better idea of it than any verbal description. It is probably the result of irritation of the posterior cervical nerves, and is most marked in cases of so-called simple basal meningitis.

DISEASES SIMULATING MENINGITIS.

Now, gentlemen, you may take it as a general rule in medicine that the more numerous are the possible symptoms of any disease the more difficult is it to diagnose that disease with certainty, for the greater is the chance of its being simulated by something else. To this rule meningitis is no exception, and you will often have great difficulty in being sure that you are dealing with it, particularly in its earlier stages. But forewarned is forearmed, and if you know what the traps are which you are most likely to fall into you will have a better chance of avoiding them. The diseases, then, for which meningitis is most apt to be mistaken are these :

1. **Typhoid Fever.**—By one form of typhoid fever—that known as the cerebro-spinal variety—meningitis may be very closely simulated indeed. All the signs of cerebral irritation—mental irritability, head retraction, photophobia, and the rest—may be present in a marked degree, and yet the case may prove to be one of typhoid after all. Not long ago, indeed, a case occurred here in which the mistake was only discovered in the post-mortem room. Pay great attention to the state of the abdomen in these doubtful cases. In typhoid it is usually full and rather tender ; in meningitis it tends to be retracted and not tender.

2. **Pneumonia,** especially, I think, apex pneumonia, may simulate meningitis very closely, particularly if head retraction be present, as it sometimes is. Careful examination of the lungs may disclose the characteristic physical signs, but even these may be absent in the first few days. The pulse-rate may help you here. In pneumonia it follows the temperature, whereas a combination of high fever with a comparatively slow pulse is much more suggestive of meningitis.

3. **Acute Gastritis** may be accompanied by many meningitic symptoms, such as high temperature, vomiting, convulsions, and constipation. The tongue here, however, is usually thickly coated, and a dose or two of calomel will probably clear everything up.

4. **Middle-ear Disease** has often been mistaken for meningitis. Indeed, in a doubtful case it is not a bad plan to puncture the membranes on chance. The simulation here is all the more puzzling, inasmuch as middle-ear disease may be followed by secondary meningitis as one of its complications.

5. **Acute Polioencephalitis** may produce a clinical picture which is the exact replica of that of meningitis. I saw such a case lately, in which it was impossible to be sure for some time what we were dealing with—vomiting, head retraction, optic neuritis, hypertonicity, all were present—but final recovery, with the exception of some spasticity of

the legs, made it extremely probable that it had been an encephalitis all along.

All of these conditions may exhibit more or less marked head retraction, but it is important to remember that this sign may be mimicked by more trivial affections. Quite recently, for example, a boy was admitted to Hanbury Ward with so much rigidity of the neck that he was thought to be suffering from tuberculous meningitis following cervical caries. Yet it eventually proved to be a case of **rheumatism** affecting the cervical muscles or some of the vertebral joints. It is also said, although I have never seen an instance of it myself, that a mere **fall on the head**—apart from any actual injury—may produce temporary head retraction in young children.

Should you find yourself puzzled in any of these ways, it is well to remember that in **lumbar puncture** you have a diagnostic aid which may save the situation. This little operation is easily performed, and, if carried out under the observance of strict asepsis, is quite devoid of risk. An anæsthetic is not really necessary. Place the patient on his face, with a pillow under the abdomen so as to bend the spine a little, and open out the spaces between the laminæ of the vertebræ. Trace the last rib on the right side back to the twelfth dorsal spine, and count down from this to the third lumbar. Place your left thumb on the third interspinous space, and enter a small trocar and cannula $\frac{1}{3}$ inch to the right of it, and pass it inwards and slightly upwards for a depth of $\frac{2}{3}$ to 1 inch, depending on the age of the patient and the thickness of his subcutaneous tissues. Withdraw the trocar, and cerebro-spinal fluid will usually exude in drops, or, if there is increased pressure, in a continuous stream. Some of this should be received into a sterilized test-tube.

There is no risk of injuring the cord, for you are altogether below its level, but the appearance of a little blood need not alarm you.

Normal cerebro-spinal fluid is perfectly clear, like distilled water. Turbidity indicates meningitis, though the opacity may be so slight that it is only noticed when the tube is shaken and held up to the light. Examination by cultures, or by merely making stained films of the deposit, may reveal the presence of the meningococcus which may be recognized in films lying inside the leucocytes (Fig. 44), or of the pneumococcus, or of one or other of the pyogenic organisms, or even of the tubercle bacillus, although in tuberculous cases the fluid is often sterile, just as it is in cases of tuberculous pleurisy.

VARIETIES OF MENINGITIS.

I may now pass on to describe very briefly the different varieties of meningitis, adopting the following provisional classification :

$$1.\ \text{Acute}\begin{cases}\text{Primary}\begin{cases}\text{Epidemic.}\\ \text{Sporadic.}\end{cases}\\ \text{Secondary}\end{cases}$$

2. Tuberculous.

3. Posterior basic (also known as 'simple basal,' and very probably only one form of sporadic cerebro-spinal meningitis).

Acute Meningitis.

Acute meningitis may either occur as a primary disease or be secondary to some other condition.

If it is **primary** it may either arise in epidemics—especially in the summer months—or may occur sporadically in isolated cases. In either case the symptoms are the same—the child is seized with high temperature, vomiting and headache, severe convulsions, and often opisthotonos supervene, and death usually results within a few hours or days. The organism responsible for the production of these cases is almost certainly the meningococcus, although it is not quite certain that the pneumococcus may not be the cause in some cases.

Secondary meningitis comes on in the course of some other—and often acute—disease, which may to a large extent mask its symptoms. It is therefore often difficult to recognize, and may, indeed, be only detected after death. I remember, for instance, having a child under my care for whooping-cough which suddenly developed a left-sided hemiplegia. Everyone who saw the case supposed, naturally enough, as there were no other marked symptoms, that a bloodvessel had given way in the brain during a paroxysm of coughing. Yet examination after death showed that we were wrong. There was no hæmorrhage, but the paralysis was due to an extensive exudation of semi-purulent lymph on the surface of one side of the cortex.

I have known the same sort of thing happen quite often in the course of a pneumonia, and it may occur also in measles or in any of the acute specifics. Not infrequently, too, meningitis is set up by suppuration in the middle ear.

In all these cases the onset of the meningeal inflammation may be very difficult to recognize, though it is perhaps oftener ushered in by convulsions than by any other sign. Its recognition is fortunately not of importance from the point of view of treatment, for you can do nothing for it; but it materially affects the prognosis, for secondary meningitis is probably always fatal, and usually within a very few days.

TUBERCULOUS MENINGITIS.

The mistake you are most likely to make in regard to tuberculous meningitis is in diagnosing it too often. One thing I would specially desire to impress upon you in this connection is that *tuberculous meningitis is very rare in the first year of life, unless as the terminal process in a general tuberculosis.* It is in the second and third years of life that apparently primary tuberculous meningitis, just like tuberculosis in general, reaches its maximum degree of frequency.

I shall not attempt to draw for you a **clinical picture** of this disease, for the reason that it would be of no real help to you if I did. At best I could only give you a sort of composite photograph, which would be very unlikely to correspond at all closely with any given case you might afterwards meet with. Tuberculous meningitis is essentially one of those diseases which can only be studied at the bedside, and by the careful observation of many cases. You will then be able to form for yourselves some idea of the extraordinary variability of the course of this affection, and of the extent to which the symptoms may depart from the classical type in one or other direction. This **classical type** was laid down by Whytt nearly one hundred and fifty years ago. He divided it into three stages—A *prodromal* stage, marked merely by peevishness, irritability of temper, and fretfulness, along, perhaps, with slight elevation of temperature, loss of appetite, constipation, and, if the child is old enough to complain of it, headache. This stage passes after a varying number of days—or even, in very insidious cases, after two or three weeks—into the *second* stage, characterized chiefly by slowness and irregularity of pulse (the typical 'cerebral pulse'), the other symptoms meanwhile becoming more pronounced. After a short duration, this is succeeded by the *third* stage, in which the pulse becomes very rapid, the temperature continues to rise, and coma supervenes.

These three stages may often be roughly traced in cases of tuberculous meningitis, but not infrequently one or more of them may be omitted, or be so transitory as to be overlooked. The individual symptom of the disease will be considered immediately, when I come to describe its differential diagnosis from posterior basic meningitis.

Posterior Basic Meningitis.

Posterior basic meningitis is so called because on examination after death the inflammatory exudation is found to be most marked over the posterior part of the base of the brain. The term, it must be confessed, however, is an unfortunate one, for the inflammation is not really *confined* to that part of the meninges, as the name might lead you to suppose, but for purely mechanical reasons that happens to be the spot where it is most easy for the inflammatory exudate—which is rather abundant in this form—to accumulate. From a strictly pathological point of view it would probably be better to speak of it simply as a form of **sporadic cerebro-spinal meningitis,** for there is every reason to suppose that the exciting cause of the inflammation is the same in both cases—viz., the meningococcus. Still, the term has received general recognition by clinicians,

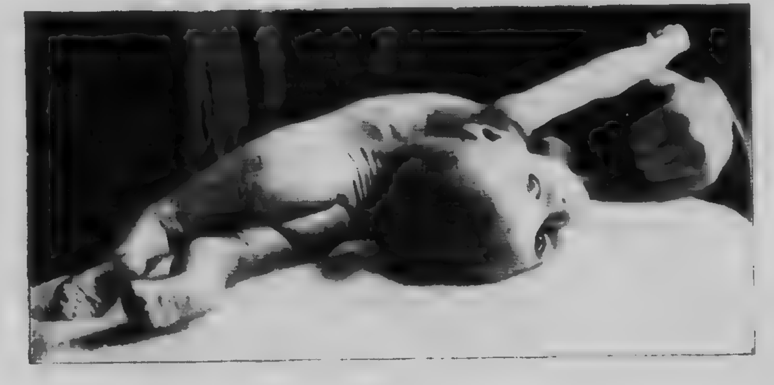

FIG. 43.

HEAD RETRACTION IN SIMPLE BASAL MENINGITIS, SHOWING 'GUN-HAMMER POSITION.'

Note also the staring eyes.

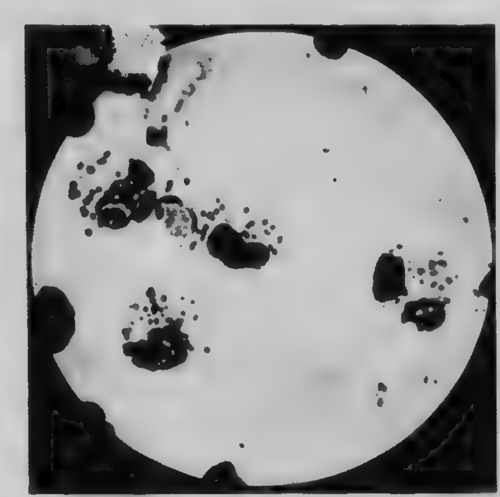

FIG. 44.

PHOTOMICROGRAPH, SHOWING THE MENINGOCOCCUS EMBEDDED IN LEUCOCYTES.

From lumbar puncture in a case of simple meningitis.

and it is as well for us to continue to use it for the present.

Posterior basic meningitis, unlike the tuberculous form, is most common in the first year of life, though it may occur at any age. This fact is so important that I wish to impress it upon you as an aid in diagnosis. It is another example of the value of what I am in the habit of speaking of as *reasonable probability in diagnosis*, which means that in a doubtful case you should lean to that disease which is most commonly met with at that age. Given, then, a case of primary meningitis in a young infant, the probability is in favour of its being the 'posterior basic' variety.

Of the **general symptoms** common to all forms of meningeal inflammation already detailed, some tend to show in this variety a peculiar prominence. Chief of these, undoubtedly, is **head retraction,** which exhibits in posterior basic meningitis a degree of development not met with in any other form. The child depicted in Fig. 43 shows this in a marked, but by no means unusual, degree, and you will recognize from the photograph the appropriateness of the description given to the attitude by French writers—viz., 'the *gun-hammer* position.' In severe cases in which **opisthotonos** is present as well, the retraction may be so extreme that the occiput almost touches the heels.

Along with head retraction, and usually proportional to it, there is **bulging of the anterior fontanelle,** which forms one of our best gauges of the increase of intracranial pressure. Whenever the intracranial pressure is high, **vomiting** is apt to set in, and it may continue with intermissions for weeks. At the same time, great emaciation is prone to occur, which is not, however, so far as one can see, the consequence of the vomiting, but is rather an essential symptom of the disease. Failure to lose weight, therefore, is always a sign that the malady is taking a favourable course.

The other signs and symptoms of this form of meningitis you will gather from the following table, in which they are contrasted with the corresponding features of the tuberculous form :

Differential Diagnosis of Posterior Basic and Tuberculous Meningitis.

Posterior Basic.	*Tuberculous.*
Common in the first year.	Rare in the first year.
Temperature may be high at first, but falls later.	Temperature rises as disease progresses.
Head retraction early, and marked.	Usually much less prominent.
Wasting usually a prominent feature.	Not marked unless in consequence of general tuberculosis.
Constipation usually absent.	Usually pronounced constipation.
Pulse and respiration not much affected.	Usually exhibit marked alterations, varying with the stage of the disease.
Eyes staring and lids retracted.	Tendency to photophobia and spasm of the lids.
No marked fundal changes.	Optic neuritis frequent.
Cerebro-spinal fluid shows the meningococcus.	Cultures sterile, or show the tubercle bacillus.

The **prognosis of posterior basic meningitis** is also better than that of the tuberculous form, for whereas the latter may be regarded as almost inevitably fatal, the former is occasionally recovered from. Unfortunately, however, recovery is by no means always complete. There is apt to be left behind as the permanent legacy of the disease a greater or less degree of **chronic hydrocephalus,** with more or less spasticity of the limbs. Many cases of hydrocephalus coming on in infancy, the cause of which cannot be traced, have probably arisen in this way. Permanent **mental impairment** of greater or less degree may also result, and the special senses be damaged. A peculiar **blindness,** without visible changes in the fundus, often lasts for quite a long time after the other symptoms have subsided, though I have never known a case in which it was permanent. On the other hand, lasting deafness may, I believe, occur.

Unfortunately, we know of no specific **treatment** for posterior basic meningitis, but you must not on that account conclude that you can do nothing for such cases. As the disease may be very protracted, careful nursing is of the first importance. If vomiting and inability to suck or swallow are persistent, nasal feeding must be carried out. Not long ago I had a case in one of the wards here in which the child had to be fed by the nose-tube for *four months*, but which finally made a perfect recovery—a result the credit for which rests mainly with the nursing-staff.

I am convinced, too, that **repeated lumbar puncture** is a therapeutic measure of great value in some of these cases, and I never hesitate to employ it wherever head retraction, bulging of the fontanelle, and repeated vomiting are prominent features. The relief to the intracranial pressure which the abstraction of one or two ounces of fluid produces in such cases is often very marked, and lasts as a rule for three or four days, when the operation can be repeated if need be.

From **drugs** I do not think you are likely to get any help, except in the treatment of special symptoms, but it is only right to say that some authorities believe in the free use of iodide of potash and mercury on the off-chance that congenital syphilis may be playing a part in the process. The later bacteriological studies of the disease, however, are rendering this chance always more remote.

Specific treatment has so far proved rather disappointing in meningococcal meningitis in children, although the results have been better in adults. In the acute stage anti-meningococcic serum may be administered,* and in the more chronic phase a vaccine, preferably prepared from cultures obtained from the cerebro-spinal fluid of the patient, may be employed; and cases have been reported in which such treatment has apparently been followed by good results. I have not myself, however, seen any case in which one could attribute recovery to the use of a vaccine, but it is right that you should give such treatment a chance.

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* Ten c.c. or more should be injected into the cerebro-spinal canal after withdrawing at least as much cerebro-spinal fluid, and the dose repeated every few days.

LECTURE XXIII

ON MENTAL DEFICIENCY IN CHILDHOOD

GENTLEMEN,—Our subject in this lecture is the mental deficiencies of children. This is a subject which deserves your very closest attention, because you will find when you go into practice that mental deficiency is not uncommon in children, and you will frequently be consulted about it. Moreover, it is a subject in regard to which if you make a mistake you are likely to get into discredit. The frequency of mental deficiency in children you will realize when I tell you that about 1 per cent. of all the children of school age are the subjects of it. But before I go further into this matter I want to define for you the **use of terms** in regard to mental deficiency. You will find, for instance, such words as idiocy, imbecility, feeble-mindedness, and backwardness all used with regard to children whose mental development is defective. At the present day the use of the first three of these terms has been practically discontinued, and the expression 'mental deficiency' is used in place of them. But if you are going to adhere still to the use of the term 'idiocy,' one would reserve it for the lowest grade of case—that is to say, those in which the mental impairment is of the extremest form. Above those, somewhat better in their mental development, will come imbeciles, and higher still, nearer to the normal children, will come those who can be called the feeble-minded. Backwardness is a thing to be sharply distinguished from mental deficiency, and therefore we shall still retain that term, and I shall define what is meant by it later on.

Coming closer to our subject, it will be convenient to look, first, at the general diagnosis of mental deficiency ;

secondly, at the clinical classification of cases of this affection; thirdly, at the etiology so far as we know it; fourthly, at the prognosis; and, fifthly, at its treatment. I shall begin with the diagnosis.

DIAGNOSIS OF MENTAL DEFICIENCY.

When you wish to diagnose a mental condition, even in an adult person, it is well to put to yourself three questions: What does the patient say? What does he do? What does he look like? In diagnosing mental deficiency in children, you will find that these three modes of attacking the problem will be of great service to you.

First, then, what does the patient say? Speech, of course, is the great mode of intellectual expression; it is one of the things by which you can judge best of a man's intellectual development. In the case of children who are mentally deficient, you will find often that it is not so much what the patient says as what he does *not* say which is helpful to you, and which will arouse your suspicion. You will remember that by the age of three a child ought to have learnt to talk fairly well; and if a child is brought to you at that age who is still **unable to talk,** you may conclude either that you are dealing with a condition of mental deficiency, or that the patient is deaf. There is a third possibility, but so uncommon as to be scarcely worth considering—namely, that the child may be the subject of what is called 'congenital aphasia.' But that is very rare. So practically a child of three years of age who cannot talk is either deaf or mentally defective. As regards what one may call *qualitative* alterations of speech, I would only point out that you must not conclude that such peculiarities are necessarily signs of mental deficiency. Take such a thing as *stammering.* That is by no means an indication of mental weakness; indeed, stammerers are often above the average of intellectual development. There is another condition, termed *idioglossia,* in which the child talks in a sort of gibberish of his own, which is entirely unintelligible to other people; and that also, although you may think it is an indication of mental deficiency, is not so. These children are often otherwise intellectually sound. On the other hand, the condition of speech called *lalling* is certainly suspicious of mental deficiency, and another peculiarity certainly exhibited by some defective children is *repetition ;* they have a habit of repeating after you the question which you ask them or the things which you say.

Next, let us consider what the child *does* which arouses your suspicions. I would point out that one great sign of mental deficiency is an **inability to perform** at the proper times complicated **co-ordinated movements.** The child will therefore be late in passing many of those 'milestones of development' of which I spoke in one of the early lectures. He will be late in holding up his head ; instead of being able to co-ordinate the muscles which hold the head on the trunk by the third month, he may not be able to do so until he is a year old, or possibly more. He will be late also in sitting up ; instead of being able to sit up at the end of the sixth or the ninth month it will perhaps not be until the eighteenth month that he can do so. The child will also be backward in walking, and, as we have just seen, in performing those co-ordinated movements which give rise to speech. Further, mentally deficient children are apt to fail in the power of grasping things ; they are wanting in the power of *acquisitiveness*, as it has been said, for grasping involves complicated co-ordinated movements by the muscles of the arms, and so you are not surprised to find that it is a function which is acquired unusually late by children whose cerebral development is defective. There will also be a lack of co-ordination in the muscles which move the eyeball and which enable the child to look fixedly at any object. Hence the gaze is vacant.

Amongst positive motor signs which will arouse your suspicion is the performance of rhythmic movements. These are a common indication of mental deficiency. For instance, there are swaying movements of the body, jerking of the shoulders, or grimacing movements. Such movements as those, constantly repeated, should arouse your suspicion with regard to the mental condition of your patient. There may also be certain tricks of movement which are suspicious. I refer to such things as hand-sucking and the making of meaningless gestures. Great restlessness is also a common sign. This often goes with sleeplessness at night, and that again with apparently causeless screaming or crying.

We now come to what the patient *looks like*. The **facies** or *facial expression* of the child is, after all, perhaps the most valuable index which you have of the mental state. I told you that the face in children is a mirror of the mind in a way that it is not in grown-up people, and there is no class of case in which that is of greater service to you than in the mentally defective. So much is this the case that many forms of mental deficiency in children have an absolutely characteristic facies, so that you can say at once, 'This child is mentally deficient, and belongs to such and such a class.' We shall see immediately when we speak of classification that one makes large use of this in the subdivision of cases of mental deficiency into different clinical groups. But, apart from those which have a characteristic facies, you will find in almost all mentally deficient children *something* in the expression which will arouse your suspicion. There is a vacant look, or perhaps grimacing, or an inability to recognize the mother or the nurse. All these things should put you on your guard. You will also find that the size and shape of the *skull* is often of great service to you in enabling you to arrive at a diagnosis, because, just as in facial expression, so with regard to the skull, there are abnormalities which are absolutely characteristic. Then you will find, in looking more carefully at such children, that you are apt to get what are spoken of as the **physical stigmata** of intellectual defect, which have been described by Dr. Warner under what he terms 'the law of coincident development.' This simply means that if one part of the body is defectively developed, other parts are apt to suffer also. If, for instance, the child has a badly-developed brain, he is more likely than other children to have congenital lesions of the heart, or supernumerary digits. Amongst the physical stigmata of mental deficiency are what are termed marked epicanthic folds—that is to say, folds of skin which come down from the upper eyelid to the lower, and cover over the caruncle. A higharched or Gothic palate is another common physical stigma, and it frequently goes with narrowness of the naso-pharynx and bad development of the jaws, and therefore also of the teeth. Then you will find adherent lobes of the ears more commonly in mentally deficient children than in others, the lobe being tacked on to the skin of the cheek instead of being left free. You find, too, not uncommonly, harelip, cleft-palate, and opacities of the media of the eyes. In a doubtful case you will look for such physical stigmata, and the finding of them will be an additional point in favour of the view that the child is mentally defective.

CLASSIFICATION OF CASES.

We next come to the second division of the subject—namely, the *clinical classification* of cases of mental deficiency in children. One would prefer to use a pathological classification if that were possible, but at present it is not. The pathology of many cases of mental deficiency is still quite obscure, and you will find it, as a practical matter, more convenient to group the cases according to the chief clinical symptoms and features which they exhibit. We shall begin with those cases which present a marked facies.

1. The first group of those are the **cretins.** It is extremely important that you should be able to recognize cretins, because they form that group of mentally defective infants which are the most amenable to treatment ; and it will depend upon your being able to make the diagnosis within the first few weeks whether the child will grow up mentally sound or whether it will be considerably defective. The recognition of a baby cretin is not always easy, and the classical pictures of cretins which you see in books are usually taken, unfortunately, from children several years old, by which time, of course, anyone can make the diagnosis ; but if you recognize them only then, you recognize them too late to do anything of service for them. In order to do a cretin real good, you have to catch him early, and if you do so, and treat him properly, he will grow up into a child who is practically indistinguishable from a healthy and normally developed one.

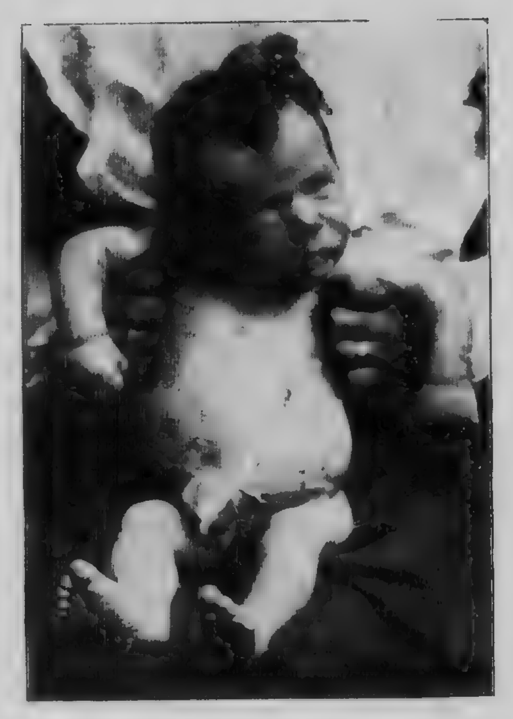


Fig. 45.

BABY CRETIN.

Note puckered forehead, puffy face, broad nose, protruding tongue, wrinkled skin, and umbilical hernia.

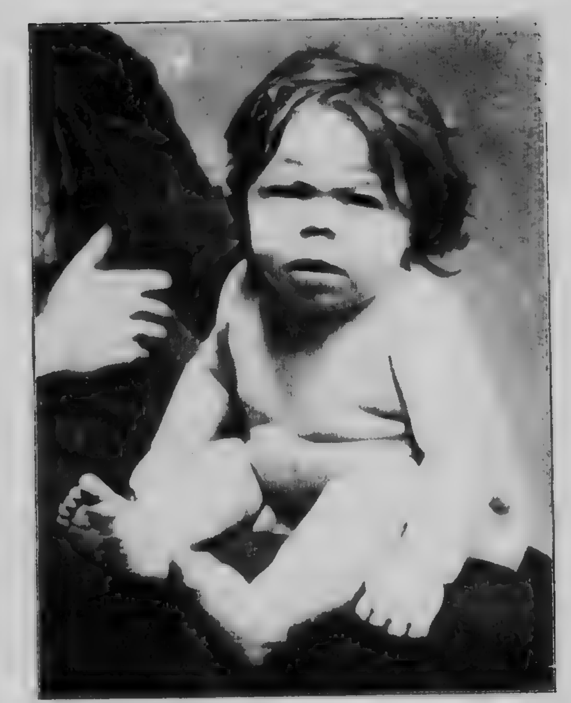


Fig. 46.

CRETIN, SHOWING CHARACTERISTIC FACIES.

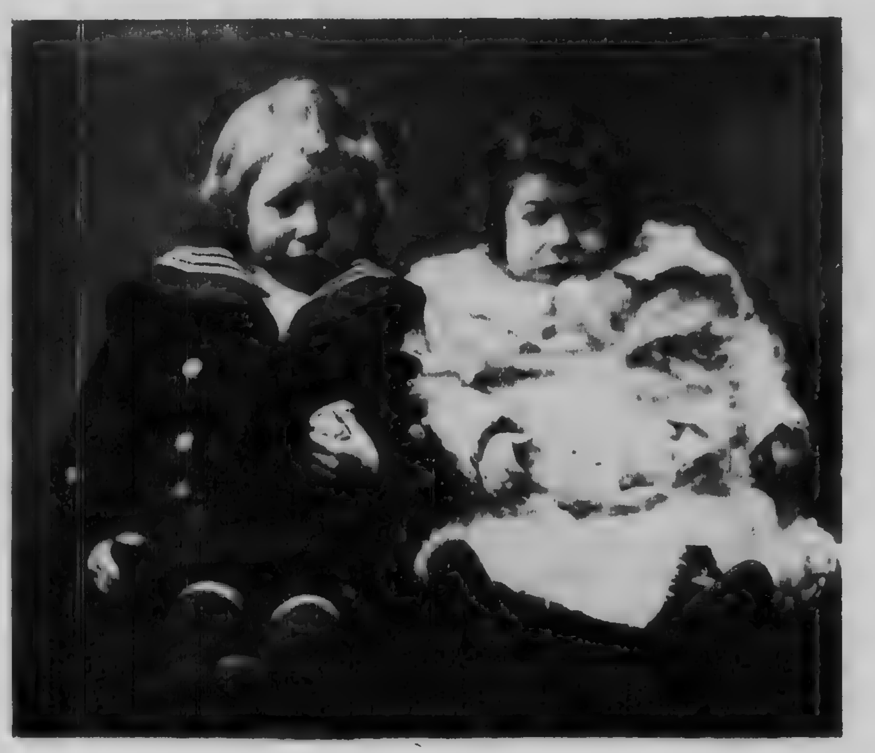


FIG. 47.

TWINS: THE GIRL A CRETIN, THE BOY HEALTHY.

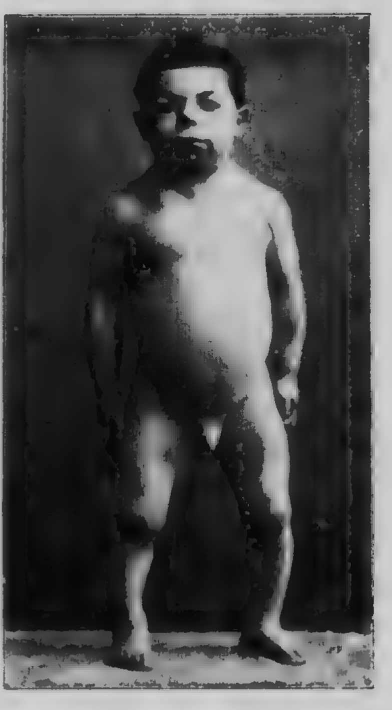


FIG. 48.

CRETIN, ABOUT PERIOD OF PUBERTY,
SHOWING 'SUPRACLAVICULAR PADS'
AND BENDING OF LEGS.

What, then, are the features by which you will recognize cretinism in its early stages ? Before I reply to this I would say that cretins are not so common as is supposed. There is too great a tendency to label almost every mentally defective child a cretin. I would impress upon you the fact that cretinism is a rare condition. Out of eighty-eight mentally deficient children of which I have notes, only 5 per cent. were examples of cretinism ; whereas Mongolism, which it is very apt to be mistaken for, occurs with much greater frequency, amounting to about 28 per cent. of my cases.

With regard to the cretin, then, the complexion is parchment-like in tint, the hair dry and scanty, and the skin redundant, so that there is a puckering of the forehead and puffiness below the eyes. There is often a squint, but by no means invariably. There is rarely, if ever, nystagmus. When you look at the shape of the head, you find it tends to be long : it is dolichocephalic. On examining the trunk you will find that the skin is dry and rough. There is frequently an umbilical hernia ; the hands are short and broad, and the finger-tips square. In manner cretins are dull and lethargic, and a feature which you will recognize in them from earliest life is that they suffer extremely from constipation. I have said nothing of the supraclavicular pads of which you will read in books, for the reason that they are rarely developed, so far as I have seen, until a cretin is at least a few years old ; they are not seen in the earliest stage, and, although it is perfectly true that you get them afterwards, yet by that time it is too late for them to be of use in diagnosis. I should, perhaps, have mentioned as a characteristic feature the tendency to protrusion of the tongue. The tongue is large, broad, and thick, and it is apt to be kept protruded. All those characteristics taken together make up a picture which, if you have once seen it, you are not likely to miss again.

2. Now I pass to consider **Mongols.** The term 'Mongolism' was first applied to this group of cases by Dr. Langdon Down, who was a physician at this hospital, from their resemblance to members of the Mongolian races—the Chinese and Japanese—in face, and particularly because

they have the oval, tilted eyes which are characteristic of these nations. When you come to compare their characteristics with those of the cretins, with which they are apt to be confused, you will find that the skin of the Mongol is smooth and white, not parchment-like in tint, and that they have a good complexion. They have no puckering of the eyebrows and no puffiness of the eyes, the hair is fine and abundant instead of being dry and scanty, and they almost always have a squint, and usually nystagmus as well. The head, instead of being dolichocephalic, is brachycephalic, and the occiput is almost in a direct line with the neck; whereas the cretin has a head projecting back beyond the neck. The finger-tips are rounded, and they have a curious incurving of the little finger. Moreover, instead of being dull and lethargic, they are bright and lively in manner. So you see they contrast in every respect with the cretins, and if you have seen a good example of each of them you need never mistake one for the other. Of the physical stigmata which I spoke of, marked epicanthic folds are a striking feature of Mongolism, and so is congenital defect of the heart. Mongols tend to suffer with great frequency from defects of the septum and other lesions of a similar sort. Out of eighteen cases of which I have notes, five had marked congenital heart disease.

3. I now pass to the third group—namely, cases of **microcephaly.** Those cases, as the name implies, are distinguished by smallness of the head. The normal head measures 13 inches in circumference at birth, 17 inches at nine months old, 18 inches at one year, and 20 inches at five years; and heads which come below these dimensions may be described as cases of microcephaly. But I want to point out to you that the characteristic of microcephaly is not merely deficiency in the circumference of the head, but an alteration in shape. I shall show you photographs presently indicating that the characteristic point about a microcephalic head is that the deficiency affects chiefly the frontal region and the vault. The base of the skull is apparently as well developed as in health. So a constricted frontal region with a small circumference are the characteristic points. The size of the head may be extremely small. The smallest

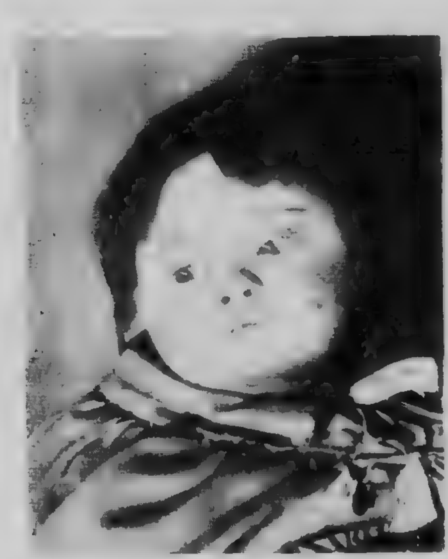


Fig. 49.

MONGOL, SHOWING CHARACTERISTIC EYES.

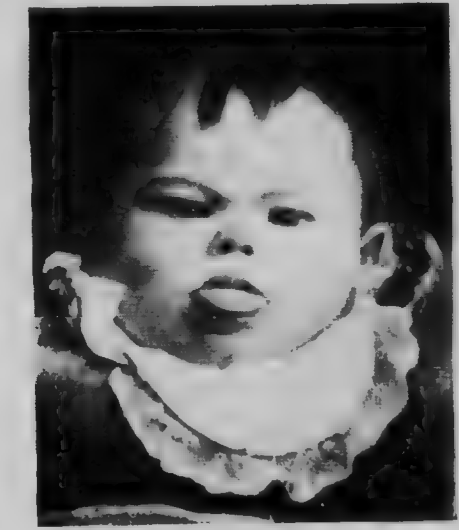


Fig. 50.

MONGOL, SHOWING PROTRUSION OF TONGUE.

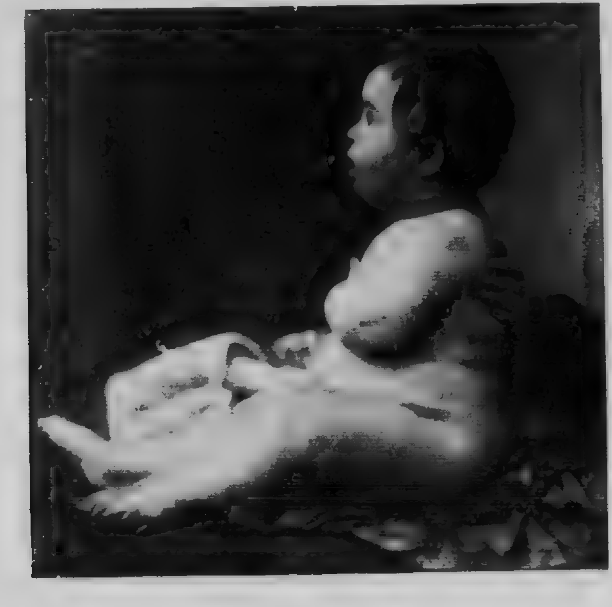


Fig. 51.

MONGOL IN PROFILE, SHOWING SHAPE OF HEAD.

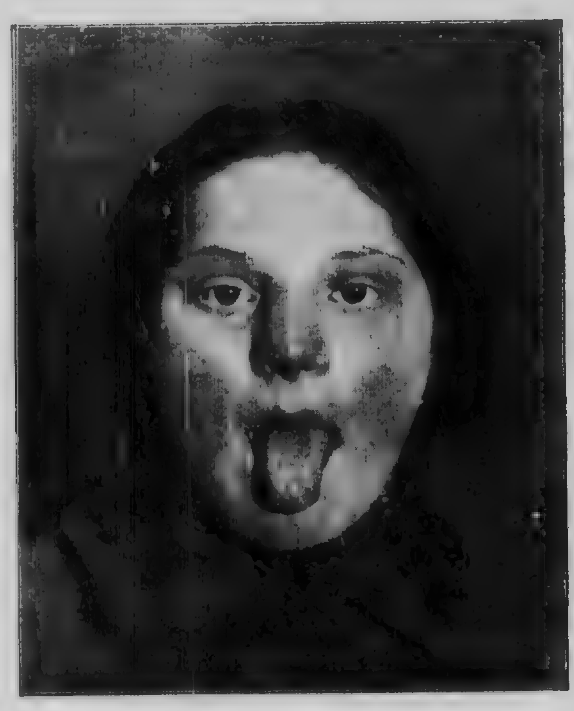


FIG. 52.

MONGOL, SHOWING FISSURING OF TONGUE WHICH APPEARS IN LATER CHILDHOOD.

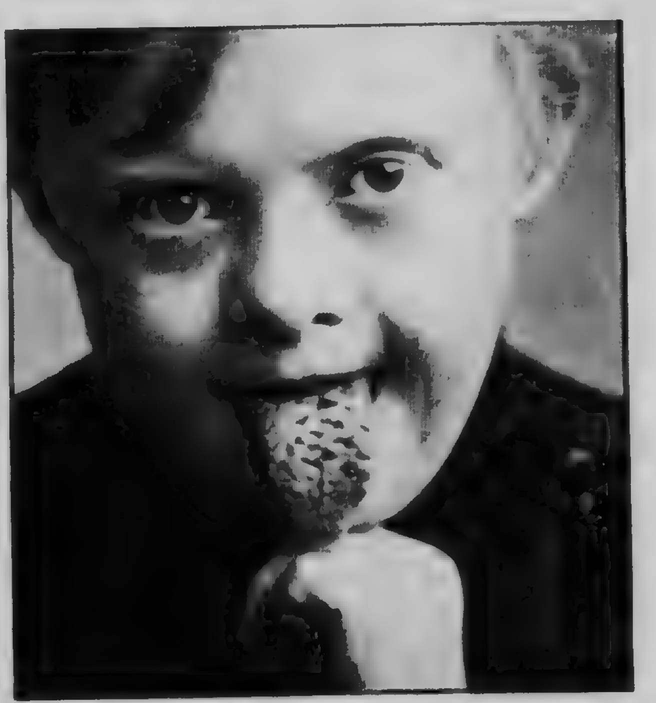


FIG. 53.

MONGOL, SHOWING FISSURED OR 'SCROTAL' TONGUE.

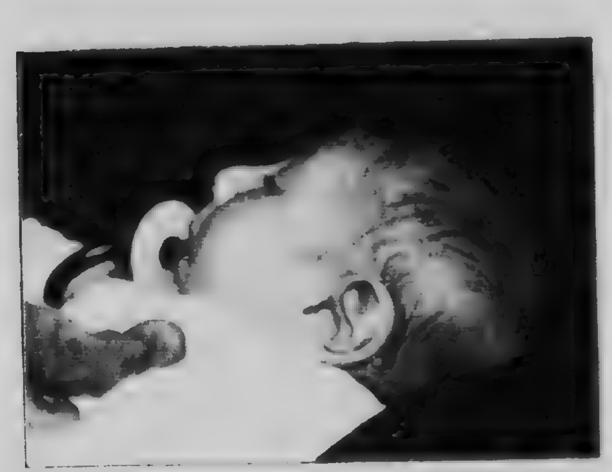


FIG. 54.

MICROCEPHALY: PROFILE VIEW, SHOWING RECEDING FOREHEAD.

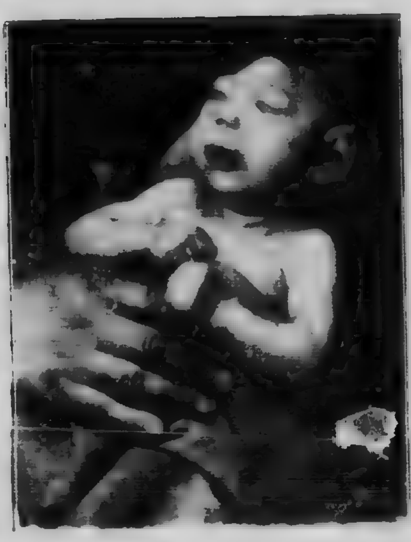


FIG. 55.

EXTREME DEGREE OF MICROCEPHALY.

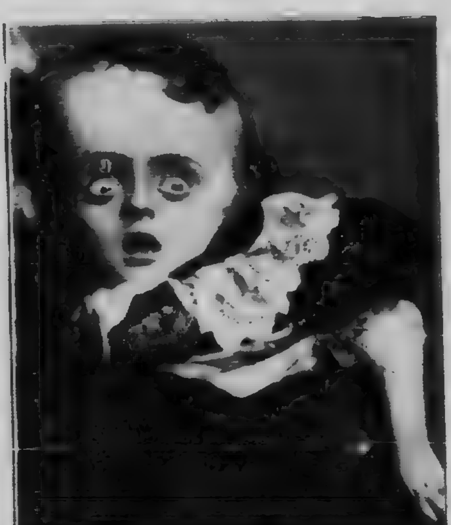
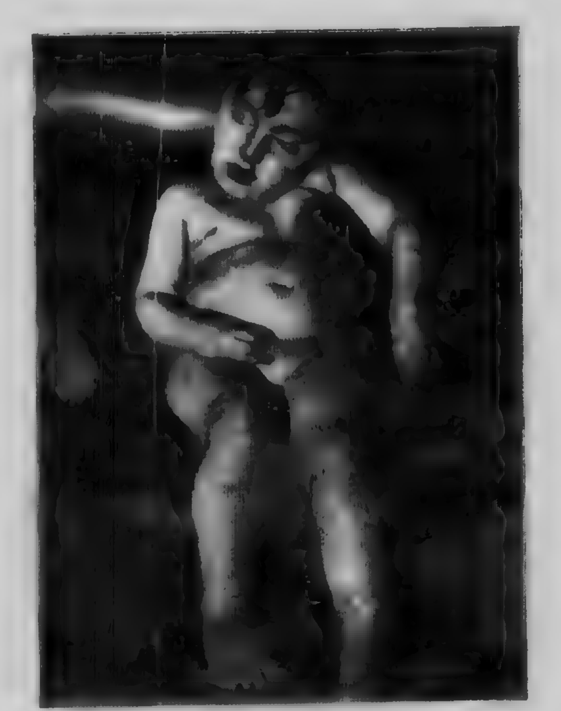


FIG. 56.

SEVERE CASE OF HYDROCEPHALUS, SHOWING CHARACTERISTIC SHAPE OF HEAD AND DEPRESSION OF EYES.



[illegible] MENTAL DEFICIENCY

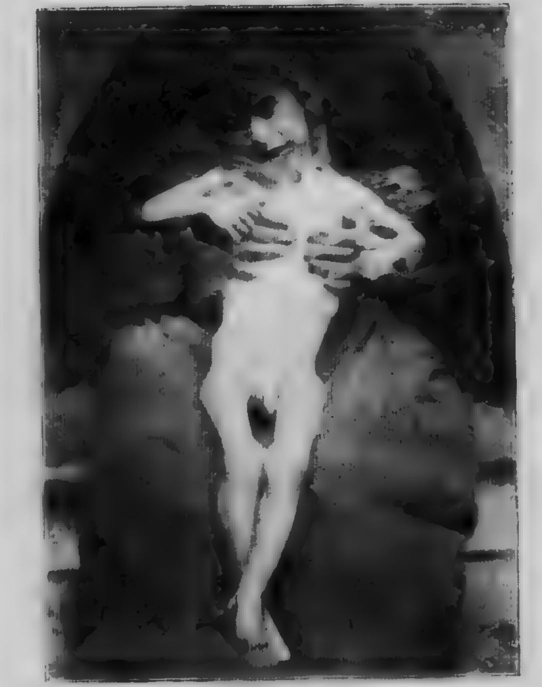


FIG. 28.

CONGENITAL [illegible]PLEGIA WITH [illegible]

I have seen was in a child three years old, in whom the total circumference was 13¾ inches—that is to say, only ¾ inch more than it should be at birth. One sees them of all measurements varying from what I have told you to nearly the normal size. Otherwise the child is usually well developed in [illegible] but has a dull demeanour and tends to be apathetic in manner.

Cases of the microcephalic type are not at all uncommon. They made up 25 per cent. of my own cases; and from what I have seen here and at Great Ormond Street Children's Hospital I think the condition tends to occur with unusual frequency in children of Jewish parents. That is quite the reverse of Mongolism, from which condition the Jewish race seems to have an immunity [illegible] than otherwise.

4. Opposed to microcephaly [illegible] **hydrocephaly,** in which the head is unusually [illegible] said much about the diagnosis of the hydrocephalic head and the means of telling it from rickets that I shall not go over that again (p. 135). Hydrocephaly is not, however, a common cause of mental deficiency, and it is extraordinary how large the head may be and yet the mental power be very well retained. The child shown in this photograph had a head 26 inches in circumference, and yet conversed quite intelligently. Such children are apt to be backward in their movements, [illegible] the large head upsets their balance and makes it difficult for them to walk; but the mental power is retained in a surprising degree, even though you know that the cortex of the brain may be flattened out almost like a sheet of note-paper.

We come next to the **paralytic group,** in which there exists some degree of paralysis in addition to the mental defect. Those cases belong to that group of infantile cerebral palsies of which I spoke in another lecture. The children who are most apt to suffer from mental deficiency with paralysis are those in whom there has been a lesion of the brain in intra-uterine life or at birth, and I think it is more common in those who have paraplegia than in those who have hemiplegia. You are apt to arrive at an exaggerated estimate of the mental defect in these children, because, owing to paralysis of muscles, they have a difficulty in getting about and living an active life like other children, and accordingly their education suffers, and to this, no doubt, part of their intellectual dulness is to be attributed.

6. There are two groups of cases of mental deficiency which are *associated with fits*, the eclamptic and the epileptic group. The distinction between these is that **eclamptic imbecility** includes those cases in which a fit has been the starting-point of the mental defect. The child lives for the first two or three years of his life quite normally, and his mental development goes on in the ordinary way; and then he has a severe convulsion which appears to damage the brain, and from that time the mental development suffers, although there is no repetition of the fit. In the **epileptic group** the case may start with a fit, but the characteristic feature is that the fits recur; the child goes on having fits, and it is in consequence of the continual fits that the mental state suffers. You may take it that if epileptic fits are frequent in a child below seven years of age, that child is almost certain to have as a consequence mental deficiency. There is nothing which apparently upsets the equilibrium of the brain more than the repeated occurrence of fits.

7. The next group is **idiocy by deprivation.** By that one means those cases which are mentally deficient because the 'avenues of knowledge' are blocked. The classical example is that of Laura Bridgman, of whom you may have heard. She was blind and deaf, and all her senses were gone, and as long as she was in that condition she was practically idiotic. Very many children suffer in their mental development because of this blocking of the avenues of information, because their hearing or their sight is bad. And whenever you are brought face to face with mental deficiency you have to ask yourself, May not this be produced by the fact that the child has a difficulty in acquiring information owing to his great inability to hear properly or see distinctly? The commonest causes are middle-ear disease, frequently the result of adenoids; and errors of refraction, particularly hypermetropia and astigmatism. As a consequence of these defects the child fails to benefit like other children from instruction at school, and so falls behind-

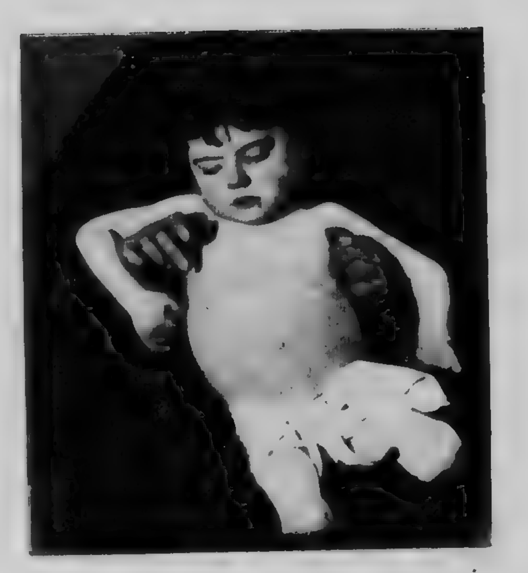


FIG. 59.

IMBECILITY OF UNCLASSIFIABLE TYPE.

Note general attitude and expression.

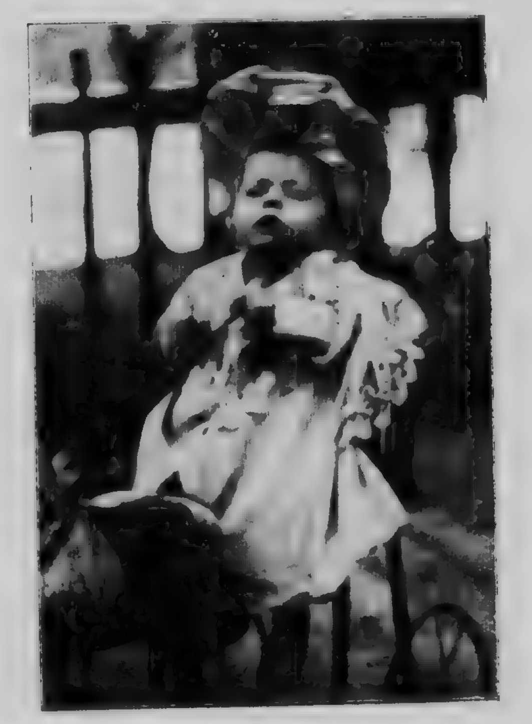


FIG. 60.

AN UNCLASSIFIABLE CASE OF SEVERE MENTAL DEFICIENCY WITH CONGENITAL PTOSIS.

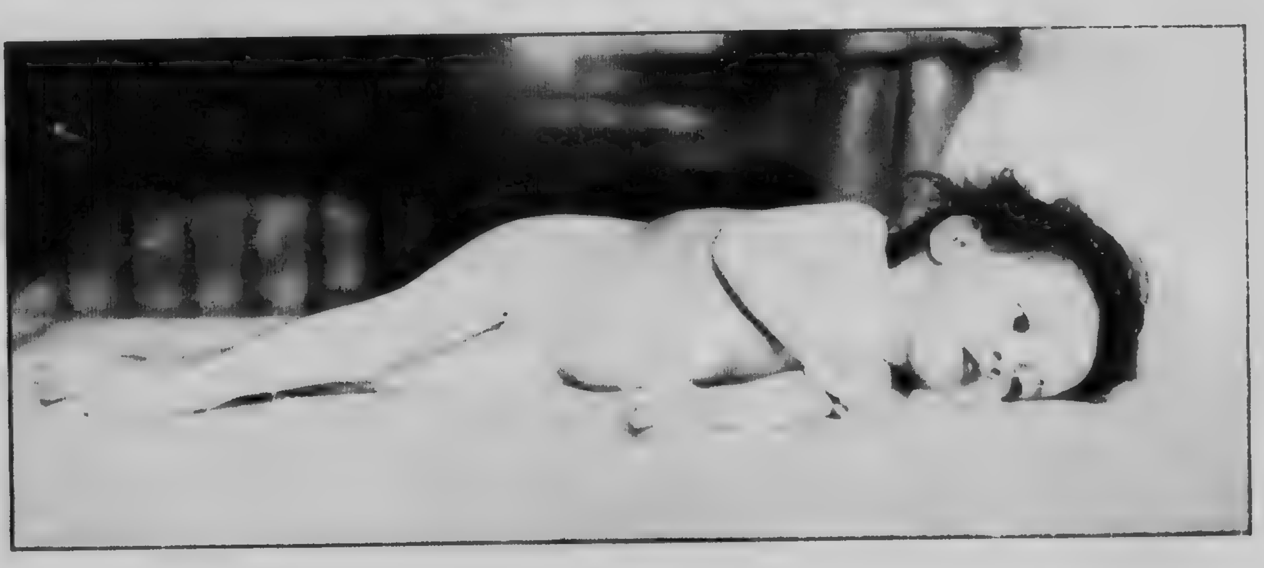


FIG. 61.

AMAUROTIC IDIOCY.

hand, and in extreme cases may be regarded as a case of mental deficiency. But many of these cases belong to the group of mere *backwardness*. And that brings me to ask what is to be your distinction between mere backwardness and true mental deficiency. You will find it convenient to remember in this connection the dictum of Charles West, *that a mentally deficient child would be abnormal for any age, whereas a backward child is merely abnormal for its own age.* For example, a backward child of six may be at a mental stage which would be normal for a child of four. But a child who is mentally deficient you cannot regard as normal for any age. You will find that useful as a practical distinction.

8. I now come to the next group, which is one of considerable rarity—namely, **primary amaurotic mental deficiency.** This is a group of cases which ought to be familiar to you here, because it was first described by Mr. Waren Tay, I think in the year 1881. It is a group which is marked by certain very definite features. First, the cases tend to occur in families, several members of a family being likely to suffer. Secondly, in all the recorded cases the children have been of Jewish parentage. Thirdly, they are characterized by the fact that the children may seem normal at birth and for a short time afterwards ; but soon loss of vision sets in, associated with characteristic changes in the fundus of the eye and with paralysis, which leads by about the second year of life to death. Such cases are very far from being common. It is well, however, for you to have them before your minds, particularly in this part of London, where we see so many Jewish patients.

9. Lastly, I come to a great group which you can refer to none of these previous divisions, and which may be described as **unclassifiable cases,** or, if you prefer a more scientific term, cases of *simple primary amentia*. They make up the majority you meet with ; they were 34 per cent. of [illegible] cases I have seen. You may call them, if you like, congenital idiocy, but I do not think that helps you much. They are characterized by mental deficiency, associated, perhaps, with some 'physical stigmata,' and you can only diagnose them by giving heed to the general rules I have

laid down—to the speech, general appearance, and behaviour of the patient—and if you attend to these points you are not likely to overlook the mental defect.

CAUSES OF MENTAL DEFICIENCY.

I now pass to consider rapidly the question of the *etiology of mental deficiency* in children. With regard to a few of these groups there is a definite causation. Cretinism, for example, is due to congenital absence of the thyroid gland, but you cannot say of many that there is such a definite causation as that. In the paralytic group, too, you have a fairly definite cause—namely, damage to the brain of some sort, occurring during the act of birth or during intra-uterine life. In the group of deprivation cases you have a definite pathology in the absence or defect of certain organs of sense. In hydrocephaly you have often a history of antecedent basal meningitis. But in regard to many of the cases you can find no definite cause at all. You will find that certain factors are commonly believed, especially by lay people, to play a part in the production of mental deficiency. You will hear it said that these children are often the offspring of intemperate parents ; or that there is insanity, or syphilis, or tuberculosis in the family ; or that the parents have been related to one another before marriage. As far as I have been able to make out, you cannot say definitely that these factors have any very great influence. Out of seventy-five cases I only found one in which consanguinity was present, and in that case the parents were first cousins. And I cannot find that the parents of mentally defective children are addicted to intemperance more than others ; whilst the coexistence of congenital syphilis is quite exceptional.

A cause which might be more likely to produce mental deficiency is bad health in the mother during her pregnancy. There have been experiments on chicks which show that if an egg is incubated at a higher temperature than usual the chick which results is apt to be abnormal and defective in development ; and you might suppose that, if a mother has an attack of fever during her pregnancy, so that the incubation of the fœtus proceeds at a temperature two or three degrees above the normal, mental defects, amongst others, might result. And I do not say that that does not sometimes happen. You will frequently get a history of bad health during the pregnancy, or a history of worry; but you must remember that such occurrences are also extremely common in pregnancies which produce normal children, and you must always allow for a tendency on the part of the mother to assign *some* cause for the mental defect in her child. I do not think it has been clearly proved that any of these things is a constant factor.

I want to speak more definitely with regard to two of the above groups—namely, microcephaly and Mongolism. The pathology of microcephaly seems to be an arrest in the development of the brain as a whole. It is not due, as has been sometimes said, to a premature closure of the cranial sutures. You know that a method of treatment was based upon that assumption—namely, craniotomy—artificial openings being made in the skull, under the impression that the brain ceased to develop because the sutures closed too soon. As a matter of fact, the reverse seems to be the case; the skull closes soon because the brain is not properly developed, so that the operation was foredoomed to failure.

With regard to Mongolism, I think you can trace a fairly definite cause. You will find that Mongols tend to be the last children of large families. It is a condition which results when the reproductive powers are exhausted, especially in the mother. Out of fifteen of my cases of Mongolism the mother was forty years old or upwards in nine, and in another three the mothers were between the ages of thirty-eight and forty. Dr. Still, of Great Ormond Street Hospital, reports eighteen consecutive cases, out of which ten were the last children of a large family, and in the others the child was the last but one. Why exhaustion of the reproductive power should result in Mongolism I do not know, but there seems to be an arrest at some stage, beyond which it is impossible to carry the development.

PROGNOSIS.

With regard to the *prognosis* in mental deficiency, you have to consider it, first, in relation to the life of the child, and, secondly, in relation to its mental progress. As regards life, you will find that mentally defective children tend to be physically weak and wanting in resisting power. It is, perhaps, fortunate that that is so, because the majority of them never reach the age of puberty. If they have an illness they take it badly, and are apt to go down before it. This feebleness is perhaps more marked in Mongols and cretins than it is in others, and the members of this group are particularly liable to be carried off by tuberculosis. You have also to remember in giving your forecast that many of them will be apt to suffer from fits, particularly in the paralytic cases. Of course, those which are epileptic already will always have fits as one of their prominent symptoms. With regard to mental development, there is nothing more difficult than to speak confidently about the future, and I advise you to be always very cautious. You may say of cretins that if thyroid gland is administered from the first weeks of life they will grow up to be almost normal individuals. You can say with regard to Mongols that in the highest grade they will be fit for physical work, but they will never be able to do much brain work of any sort, and will always lack initiative. The prognosis of microcephaly will depend on the size of the skull; the smaller the skull the more hopelessly idiotic the child will be, and it is only in those children in whom the skull is near the normal size that you will have any appreciable mental power at all. With regard to the epileptic cases, you may assert that they tend to get worse rather than better as they go on: each successive fit leaves the brain in a more damaged condition than it was in before. But the great difficulty in the matter of prognosis is in regard to those unclassifiable cases which make up the last group. It will be well to remember, as has been pointed out by many observers, that the more healthy and natural-looking the child is, the worse is the prognosis as regards mental development. Those cases which look best in physical development often give the worst outlook with regard to mental improvement. As Dr. Langdon Down has said, the prognosis is hopeful, contrary to what is often thought, 'inversely as the child is comely, fair to look upon, and winsome.'

TREATMENT OF MENTAL DEFICIENCY.

The *treatment* I can dismiss in a few words, because, I am sorry to say, for many of them you can do but little. I urge you always to remember, however, that there is a physical side to mental deficiency, and although you can do nothing for the mental development of these children, you can do much for their physical state ; you can remove obstacles out of the way of the feeble brain. You can remove adenoids, which make it difficult for them to hear. You can order them spectacles, and make it easy for them to see. You can divide tendons if they are paralyzed, and make it easier for them to use their limbs. If the child is rickety, you can treat the rickets. By removing these difficulties you can, especially where there is only slight mental deficiency, make all the difference to the child, and put him upon a very much higher plane than before.

With regard to mental improvement, all you can hope to do is by careful and persistent training. Your chief duty here will be to encourage the mother to persevere, to point out to her that improvement must necessarily be very gradual, but that all depends upon her own efforts. The child must be encouraged to take notice of his surroundings, and to do little things for himself as far as possible, whilst, on the other hand, all bad habits must be steadily discouraged. Patience, especially the affectionate patience of a mother, can often work wonders in these cases. In recent years much progress has been made in the training of mentally defective children, and their lot has never been so favourable as now. Under the Elementary Education Act of 1899, special classes have been instituted under the County Council in London for the training of defective children ; and much has been done by the Kindergarten methods in teaching these children to use their muscles and make the best use of the feeble brain-power they have. It is only on those lines that you can hope to do good in those who are improvable. For those who are unimprovable there is no refuge but the idiot asylum, or, for people who are able to afford it, special guardianship.

LECTURE XXIV

SOME DISORDERS OF THE HEART IN CHILDHOOD

GENTLEMEN,—In the present lecture I propose to consider with you some of the commoner disorders of the heart met with in childhood, apart from those acute rheumatic affections which were dealt with on a previous occasion.

At the outset it may be well to remind you again of some of the **anatomical** and **physiological peculiarities of the heart** in children to which I referred in an earlier lecture (p. 10). You will remember that the heart lies naturally at a higher level in early life than it does in the adult, the apex-beat being often in the fourth space instead of the fifth. The apex-beat is also further out than it is in the adult, being beyond the mammary line up to the third year, and in it from the third till about the tenth years, after which it assumes the adult position.

You will remember also that **irregularity** of the heart is very common in early life, especially during sleep, and has not necessarily any pathological significance. It is of the nature of what is known as 'sinus arrhythmia,' and may be distinguished from the irregularity of organic disease by the fact that it disappears on exertion. The greater rapidity of action of the heart in early life should also be borne in mind, the pulse-rate being—

At birth, about 130.
In the second year, about 110.
In the third year, about 100.
In the eighth year, about 90.
In the twelfth year, about 80.

It is slightly more rapid in girls than it is in boys.

As regards the **heart sounds** in childhood, I would remind you again of the relatively greater intensity of the pulmonary than of the aortic second sound up till about the twelfth year.

In early life, too, so-called **cardio-pulmonary murmurs** are not uncommon, and are apt to lead to confusion. They may be distinguished from the systolic murmurs of organic disease by the fact that they are only audible during one phase of respiration, being usually present during expiration and disappearing during inspiration.

Displacement of the heart is not uncommon in childhood. It may be due to (1) pleuritic effusion (usually left-sided) pushing it over; (2) to fibrosis of the lung pulling it over; (3) to atelectasis, which causes displacement towards the affected side; (4) to transposition of the viscera; true dextrocardia, without displacement of the viscera, also occurs, but is very rare. You will rarely have any difficulty in determining the cause of the displacement in any case if you examine the patient carefully, but slight degrees of fibrosis of the lung causing displacement are apt to be overlooked. The X rays are of great help in determining the cause in a doubtful case.

I do not intend to say much about what is really the commonest form of heart-trouble in childhood—namely, **chronic valvular disease,** for the reason that it is in all essential respects the same as valvular disease in the adult, and calls for the same management. I would, however, give you one emphatic piece of advice, and that is, *Don't treat murmurs!* By this I mean that you are not to suppose that treatment is called for merely because you have detected a heart murmur on routine examination. So long as the lesion is fully compensated—*i.e.*, is producing no symptoms—it may safely be let alone. This simple rule, however, is constantly disregarded, and I often have children sent up to hospital here to be treated 'for heart disease' because a school doctor has found a murmur in the course of his official inspection. Great harm may be done by such excess of zeal, for not only are the parents unduly frightened, but the child is apt to be condemned to a semi-invalid life quite unnecessarily, and greatly to the impairment of his happiness and often of his education. In such a case all that is necessary is to warn the parents of the danger of fresh attacks of rheumatism, and to advise them to send the child to bed and call in medical advice so soon as even the slightest pain in the joints or limbs is complained of. At the same time, they should be told that the heart has been 'damaged,' but that this need not necessarily interfere with the child's health or prospects in life, although it will be wise to see that any overexertion is avoided, and that in deciding upon the patient's future career strenuous occupations and those which entail much exposure are avoided.

As regards prognosis in valvular disease it is well to remember that, other things being equal, it is worse the younger the child. Puberty is the period to be dreaded, for it makes great demands upon the heart, and if the lesion be a severe one there may not be sufficient reserve to meet these. Hence the outlook is better if the damage be not inflicted until the period of puberty is passed.

CONGENITAL AFFECTIONS OF THE HEART.

Congenital heart disease is the result in most—perhaps in all—cases of a malformation which originates early in intra-uterine life, but it may possibly, in a few instances, be the consequence of a fœtal endocarditis. Into these vexed pathological questions I do not propose to enter further, for the mode of origin of the lesion is really of very little clinical importance except for this, that if fœtal endocarditis *should* be the cause in any case, there is a certain risk of fresh endocarditis being grafted on the top of it during childhood. The bare possibility of this happening should be an inducement to one to guard children with congenital heart disease from infections of all sorts—rheumatic and other—as carefully as possible.

A practical question which often arises at the bedside is this: Is the murmur which is present due to **a congenital or an acquired lesion?** To this question it is often difficult—sometimes impossible—to give a satisfactory reply. In deciding it the following points have to be considered:

1. *The History.*—A clear history of previous acute rheumatism or chorea is, of course, strongly in favour of an acquired lesion; even a history of sore throats or 'growing pains' should make one suspicious.

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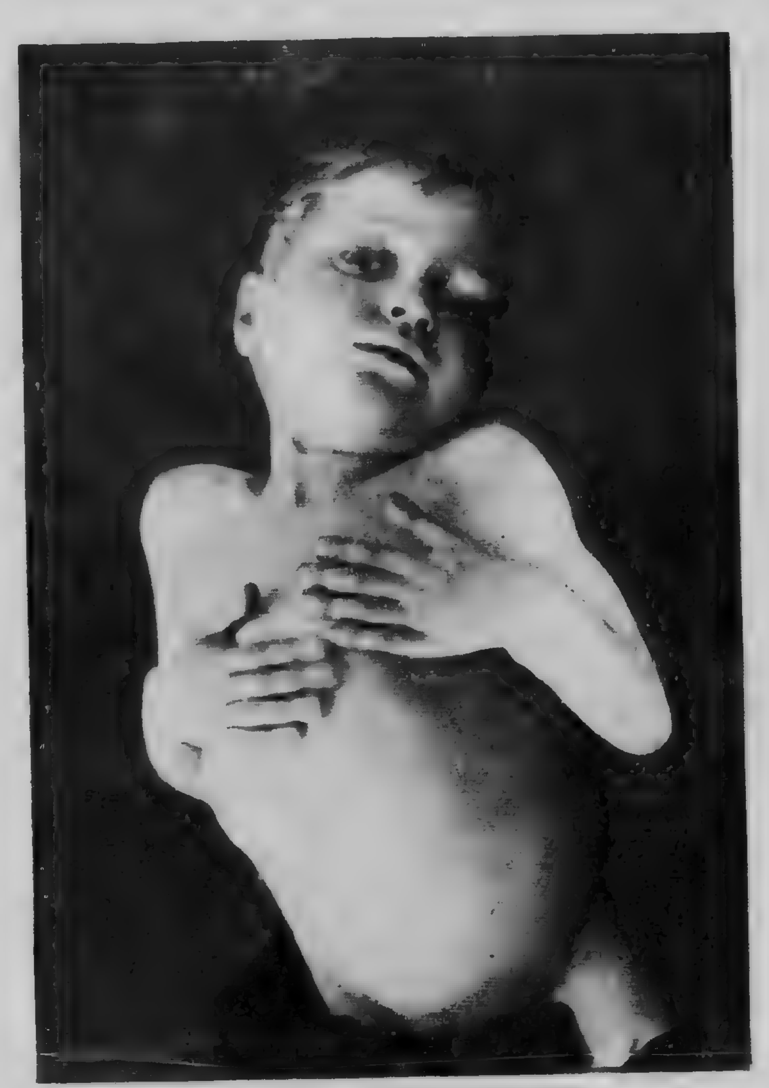


FIG. 62.

CLUBBING OF THE FINGERS IN CONGENITAL HEART DISEASE.

2. *The Age of the Patient.*—The younger the child the more likely is it that one is dealing with a congenital affection. Rheumatic endocarditis is almost unknown below the age of two, and is rare below five.

3. *Site and Character of the Murmur.*—Congenital murmurs are usually best heard *internal* to the apex or in the pulmonary area. Acquired murmurs are usually loudest at the apex-beat, and are conducted out from it. Diastolic and presystolic murmurs, wherever heard, are oftener acquired than congenital. A very loud murmur with no subjective symptoms is usually congenital.

4. *Enlargement of the left heart* is in favour of acquired, of the right heart of congenital, disease.

5. Marked *cyanosis* and the presence of *clubbing* point to a congenital lesion.

6. The *coexistence of other malformations*—*e g.*, harelip, cleft-palate, etc., is to some extent in favour of the lesion being congenital.

Even when due consideration has been given to all these points, you will sometimes be in doubt. This is specially apt to happen in the case of a child of about five years of age whose heart is being examined for the first time and whose past history is indefinite. Hæmic and cardio-pulmonary murmurs are also a fertile source of error.

Supposing that you have decided that the lesion is congenital, the next point to determine, if you can, is its exact nature. At this point it is well to remember that the four **commonest congenital lesions** in order of frequency are these: *patent ventricular septum*, *pulmonary stenosis* (these two often coexisting), *patent ductus arteriosus*, and *aortic stenosis*. The chief signs of these different lesions are briefly as follows:

1. *Patent Septum.*—A systolic bruit with its maximum intensity about the level of the fourth left costal cartilage close to the sternum: possibly cyanosis and clubbing.

2. *Pulmonary Stenosis.*—A systolic bruit, often with a thrill, having its maximum intensity in the pulmonary area; enlargement of the right ventricle: usually cyanosis and clubbing.

3. *Patent Ductus.*—A long murmur beginning with systole

and running right through into diastole, best heard in the pulmonary area; frequently a patch of dulness in the second left interspace close to the sternum; no cyanosis or clubbing.

4. *Aortic Stenosis.*—A systolic murmur *accompanied by a thrill*, loudest at the base either to the right or left of the sternum; associated with hypertrophy of the left ventricle, but not accompanied by cyanosis.

It must also be remembered that several of the above lesions may coexist or be associated with rarer ones. Patent ductus, for instance, rarely occurs alone, and pulmonary stenosis and defective ventricular septum are often present together. *Patent septum ovale*, though often diagnosed, is really, according to post-mortem evidence, a rare lesion, and still more rarely recognizable, as it does not appear to produce a murmur.

This leads me to point out that considerable malformation of the heart may exist **without the presence of any murmur** at all, cyanosis and clubbing of the fingers being the only objective signs of any abnormality.

When all is said and done, then, you will see that the diagnosis of the exact nature of the lesion in a case of congenital heart disease is often mainly a matter of guesswork, and it is perhaps better not to bother much about it at all, but to content yourselves with dividing cases into two groups —those with cyanosis and those without. For practical purposes I am inclined to think that this is quite sufficient.

One other point in diagnosis must be mentioned. You must not suppose that all cyanosed babies suffer from congenital heart disease. Every now and then you will meet with a case—usually within the first few weeks of life—in which the child is noticed to 'go black' at times, especially about the lips and in the hands and feet. This is particularly apt to happen after the bath or on exposure to cold, and is commonest in premature infants. It is, I think, a **vaso-motor disorder,** analogous to Raynaud's disease, and tends to pass off after a few months as the child gets stronger.

Having diagnosed the existence of a congenital lesion, the next point you have to consider is the **prognosis,** and you are sure to be asked: Can the condition disappear? Is it serious? What are the child's chances of life?

As regards the first of these questions—the **possibility of recovery** from the lesion—you are justified in the vast majority of cases in replying in the negative. Notwithstanding this, however, I have met one or two cases in which a congenital systolic murmur, not accompanied by cyanosis, finally disappeared. Presumably these were examples of a slight defect of the septum, which gradually closed, but you can rarely count on this happening. Your reply to the second question will depend upon the degree of cyanosis present: roughly speaking, the more marked the cyanosis the more serious the lesion.

As regards the chance of life in congenital heart disease, you may say that about half of all cases—cyanosed or not—die before the fifth year, and that, of the cyanosed cases especially, few reach adult life. They die either of intercurrent disease—especially pulmonary affections, which they stand badly—of hæmoptysis, or of more or less sudden syncope (often attended by convulsions). Should they survive, they are apt to be stunted in development, and, in cases of pulmonary stenosis especially, pulmonary phthisis is prone to develop in adult life. Much, of course, will depend upon the amount of care which can be taken of the child, but even in the most favourable circumstances survival to middle life is exceptional. Some lesions, however, such as aortic stenosis and uncomplicated patent ductus, offer a better chance than others.

Congenital heart disease offers little scope for **treatment.** The lesion is in its nature incurable, and all you can do is to try to mitigate its effects. The patient should therefore lead as sheltered a life as possible, all overexertion, physical and mental, being avoided. You will judge of what it is safe to allow the child to do by its effect on the cyanosis; anything which increases the latter is harmful. Cold also is injurious in these cases, and all exposure to infections should be guarded against with the utmost care. The diet should be so arranged as to lessen as far as possible the occurrence of flatulence, which is apt to be present in these cases, and adds materially to the embarrassment of the heart.

Drugs are of little help, and the ordinary heart tonics, such as digitalis, of none at all. For the attacks of palpitation, collapse, and cardiac distress which are apt to occur from time to time, the occasional use of a stimulating carminative mixture—*e.g.*, ammonia and ether—is of service, and when the right heart becomes seriously overdistended, cautious blood-letting by means of leeches may be of temporary utility.

FUNCTIONAL DISORDERS.

Considering the highly sensitive condition of the nervous system in children, it is perhaps surprising that they are not more subject to functional disorders of the heart of nervous origin than experience shows to be the case. Functional **palpitation,** however, is not uncommon in children of school age, and is usually due to flatulent indigestion. It resembles in its characters the same condition as seen in the adult, and its treatment consists in attention to the digestion and the administration of bromides and carminatives.

Arrhythmia, as I pointed out when speaking of the physiological peculiarities of the heart in childhood, occurs very readily in early life, and is of no significance. It has the characters of a **sinus arrhythmia**—*i.e.*, the variation is in rate, but not in force, and often follows the phases of respiration. One also meets not unfrequently with cases of irregularity due to the occurrence of **extra systoles,** and in this sort of case attacks of palpitation as well are prone to appear at intervals. Flatulence is no doubt at the root of both. It is characteristic of these—and, indeed, of all functional arrhythmias—that the heart tends to become steadier on exertion—indeed, it often becomes quite regular—and whenever you find this happen, you need have no real anxiety about the condition. The treatment of this form of irregularity is the same as that of palpitation.

Cases of extreme irregularity of the heart occasionally turn up in which it is very difficult to say whether one is dealing with a functional or an organic condition. I remember, for instance, a little girl, six years of age, being brought to me for attacks of pallor and palpitation which were said to come on upon exertion. I found that she was slightly anæmic, and had an extremely irregular heart, but without any sign of enlargement and showing no murmur. Treatment by bromides had little effect, but as soon as she was put on digitalis, her heart became perfectly regular.

From the character of the irregularity in this case and the promptitude with which it subsided under digitalis, I think there can be no doubt that one was dealing with a case of **auricular fibrillation**; but if so it seemed to be of nervous origin, for there was no sign of any organic disease in the heart, and no history of rheumatism nor overstrain, nor of anything which could have damaged the myocardium. I believe, however, that the heart experts are not yet quite sure whether auricular fibrillation is ever of purely functional nature. Be that as it may, there can be no doubt that you will occasionally meet with cases of arrhythmia in childhood in which there is no sign of organic disease, and which yet respond in the most dramatic manner to the administration of digitalis.

SUSPECTED HEARTS.

You will meet occasionally in practice with a type of heart which will cause you a good deal of uncertainty and anxiety both in diagnosis and prognosis. I refer to cases in which a child suffers from symptoms which raise a suspicion of cardiac disease, but in which the physical signs are equivocal. These we may speak of for convenience as 'suspected hearts,' and this is the sort of case I mean. A child of school age (for these cases are not met with in infancy) complains of undue fatigue on exertion, perhaps with slight breathlessness, and possibly also of some discomfort or actual pain about the precordia. It is noticed that he looks pale or 'gray' at times, and occasionally may actually seem to be going to faint. At the same time he is languid, depressed, uninterested in work or play, and his weight tends to go down.

In the course of the routine physical examination of such a patient it is found that the apex-beat is a little further out than it should be, and the first sound in the mitral area is unusually short, and may show some degree of impurity, or even the presence of a short systolic murmur; whilst the pulmonary second sound tends to be accentuated. The pulse also may be rather rapid, and is abnormally responsive to effort or to change of position. The artery wall also may be more easily palpable than it should be at this age.

Now, what does this mean? Is there, as the parents will ask, any disease of the heart, and is it serious?

It is really rather difficult to be sure of the exact state of things in such a case as I have described, but I think the probability is that one is dealing with some loss of tone in the heart-wall which has resulted in slight dilatation of the left ventricle. Some children appear to have by nature a heart which is deficient in tonicity, and if any strain be thrown upon it—either in consequence of the demands made upon the organ during rapid growth, or as the result of overexertion in games—it yields. Or, on the other hand, the tonicity may have been normal to start with, but it becomes impaired as the result of poisoning of the myocardium by the toxins of certain acute specific fevers—influenza and pneumonia appear to be the commonest—or the heart muscle is damaged by high fever, with the result in either case that slight dilatation takes place.

Such cases, of course, are not in any real sense serious in that there is no immediate danger to life, and with care the heart may recover completely, but, on the other hand, the health and activity of the child may be considerably interfered with for some time, and the heart may even be left permanently in a state of impaired efficiency and subject to more acute dilatation on moderate provocation. They will, therefore, cause a good deal of anxiety.

In the management of these cases it is difficult to do enough without doing too much. One wants to give the heart every chance of recovering itself without at the same time interfering more than is absolutely necessary with the child's education; and in the case of a boy at a public school, particularly, the reconcilement of these conflicting objects is often a difficult task. In the most marked cases, and where the damage to the heart is of recent origin, a spell of bed is no doubt the best plan, but how long the patient must remain there you can only determine by experiment, noting carefully the effect upon the heart and pulse of the resumption of the vertical position and of any degree of activity.

In the milder and more chronic cases it will be sufficient to restrict the amount of exertion undertaken to walking on the level, and perhaps to the quieter forms of games, and to insist upon the patient lying down for an hour or two in the middle of each day, whilst in the slightest cases of all the latter rule may safely be relaxed. It will readily be understood, however, that even such slight restrictions as these may make it impossible for a boy to remain at a boarding school.

As regards the rest of the treatment I believe ample hours of sleep are very important in such cases—a point which is not always attended to at schools. Drugs are of secondary importance, but tonics such as iron and strychnia are useful, and, in the severer cases at least, digitalis may be of service.

Complete recovery may be a slow process, requiring months, or even years, of care to bring about, but throughout the whole period of treatment you must keep before yourself the object of throwing no undue strain upon the heart, whilst avoiding so far as possible making the patient an invalid.

PERICARDIAL EFFUSION.

Acute rheumatic pericarditis is rarely attended by much effusion, and the marked extension of the area of heart dulness which so often goes along with it is not due, as is often supposed, to effusion, but to dilatation of the heart itself. The real effusions into the pericardium in childhood are nearly always **purulent,** and the great majority are met with in children of less than four years—an age at which, as I have already told you, acute rheumatism is almost unknown. The pneumococcus is the commonest exciting cause of the effusion, and the pericarditis is usually only an incident in a general pneumococcal septicæmia which has followed upon an attack of pneumonia ; and an empyema or pneumococcal peritonitis commonly occurs along with the pericarditis. Very rarely has one to deal with a **primary pyocardium** in which there is no suppuration elsewhere. The following is an example of such a case :

A female child, eight months old, was admitted to hospital with a history of having been taken acutely ill ten days previously with vomiting and general distress. She was a well-nourished baby, but looked ill, with laboured and rapid respiration, a very rapid pulse, and slight cyanosis. The temperature was normal. On examination, the area of superficial heart dulness was found to extend up as high as the second left costal cartilage, and outwards beyond the mammary line. *Resistance on percussion over the heart was markedly increased.* The sounds were very feebly heard, and there was no friction sound. The lungs were clear. On examination of the abdomen, the left lobe of the liver was found to be pushed down to a considerable extent.

A diagnosis of pericardial effusion was made, and a needle, inserted just outside the apex-beat, drew off pus. Under an anaesthetic the pericardium was incised close to the left edge of the sternum, and 12 ounces of pus evacuated, which contained numerous Gram-positive lanceolate diplococci.

The patient was greatly relieved at first, and the wound discharged freely, but on the fourth day the temperature rose, and the child suddenly collapsed without apparent reason. At the autopsy some masses of purulent lymph were found in the pericardium, but all the other organs were healthy.

The case I have just quoted illustrates very well the chief signs by which you will recognize the existence of pericardial effusion—viz., extension of the area of heart dulness in every direction, but especially upwards, with increased resistance on percussion ; feeble or absent heart sounds and displacement downwards of the left lobe of the liver. If there be—as there unfortunately often is—some consolidation of the left lung or a left-sided empyema, the diagnosis is much more difficult, and may, indeed, be almost impossible. In such a case the use of the X rays may help. Except for purposes of prognosis, however, the establishment of the diagnosis is of doubtful advantage to your patient except in those very rare cases, such as the one described, where the pyo-pericardium is the sole lesion, and where, if the diagnosis be made in time, there is at least a chance of saving the child by operation. In those unfortunately far more numerous instances in which there are foci of suppuration elsewhere, and in which there has been a general pneumococcal infection, or in which there is an empyema or extensive pneumonia, you will rarely be able to do much to help the patient.

LECTURE XXV

THE BLOOD DISORDERS OF EARLY LIFE

GENTLEMEN,—I need not emphasize the importance of healthy blood to a young child. The blood is the medium by which all the processes of nutrition are carried on, and if it is deficient in any respect growth and development must inevitably suffer. For it is not a matter of indifference whether, during the early years of existence, when the tissues are as wax, to be moulded in one direction or another, the cells are bathed in rich blood or in a poor and watery substitute for it. Throughout the whole of life the body may exhibit a bias towards health and strength or towards delicacy and feebleness, according as the blood was normal or not throughout these impressionable years. You will readily understand, therefore, how important it is that you should become acquainted with the chief causes of anæmia in infancy in order that you may be in a position to do all that you can towards warding them off.

FORMATION OF THE BLOOD IN INFANCY AND ITS PECULIARITIES.

Before proceeding further, however, it is necessary that you should be made acquainted with the peculiarities of the blood in early life and its mode of formation.

The infant embarks upon his extra-uterine career possessed of a **number of red cells** considerably in excess of that of the adult, the total being about 5½ millions per cubic millimetre. The amount of **hæmoglobin** is proportionately high, being about 110 per cent. The meaning of this surplus of red cells and hæmoglobin I cannot discuss with you now, but I would only point out that it is very short-lived, and by the end of the second week the adult figure of 5,000,000 red cells is reached, and persists throughout life. The

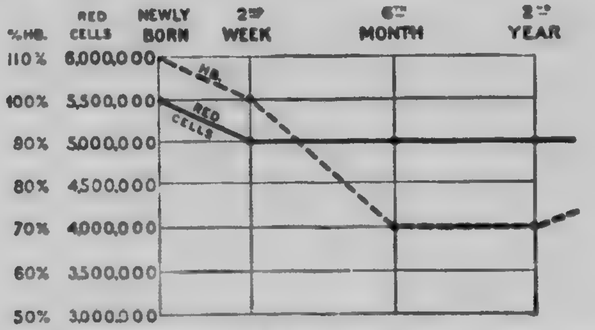


DIAGRAM 10.—PROPORTION OF HÆMOGLOBIN AND RED CORPUSCLES THROUGHOUT INFANCY.

hæmoglobin falls even more markedly, and the fall continues after that of the red cells has ceased, till by the sixth month the normal percentage for infancy (70 per cent.) is reached.

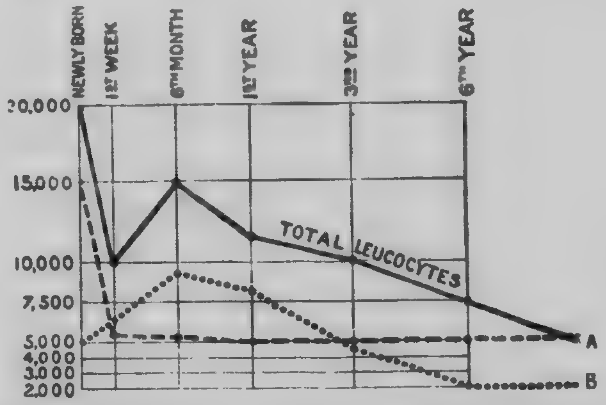


DIAGRAM 11.—ABSOLUTE NUMBER OF LEUCOCYTES PER CUBIC MILLIMETRE AT DIFFERENT AGES. A, POLYNUCLEARS; B, LYMPHOCYTES.

This low figure persists till the second year, when a gradual rise makes its appearance, and continues till about the age of puberty.

The rapid reduction in the red cells and hæmoglobin in the early weeks of life has this special clinical interest, that it is probably the cause of that familiar phenomenon, **icterus neonatorum,** the products of destruction of the red corpuscles rendering the bile so viscid that it stagnates in the liver, and is reabsorbed, with consequent jaundice.

In infancy, as in the adult, the red cells are formed exclusively in the red bone-marrow. The **extent of the red marrow** is very different, however, in the two cases. In the young child the marrow is red throughout the whole length of the long bones, and it is only as the growth of the bones becomes completed that it retreats to their extremities, which are the only parts of the long bone in which such marrow is found in the adult. One consequence of this must be that the child has no great reserve capacity to form red cells as the adult has, for there is no yellow marrow for the red to encroach upon when a greater demand for coloured corpuscles arises. Some of the results of this we shall see later. The **white cells** in the child resemble closely in their histological characters the corresponding cells of adult life, but differ markedly in their relative and absolute numbers.

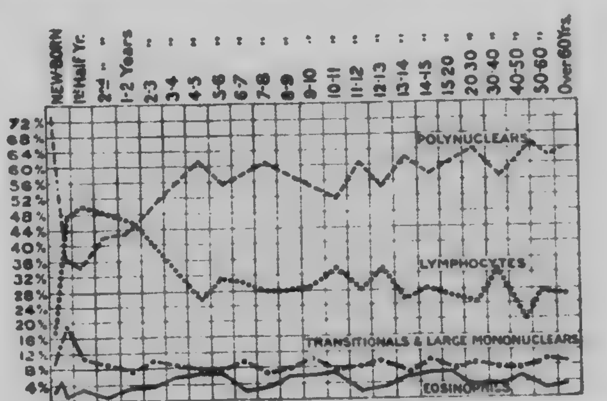


DIAGRAM 12.—DIFFERENTIAL PERCENTAGE COUNTS THROUGHOUT LIFE. (AFTER CARSTANJEN.)

The total number of white cells in the blood at birth is about 15,000 per cubic millimetre; by the end of the first year it has sunk to about 14,000, by the second year to 12,000, and by the third to 10,000, after which the decline goes on steadily till the usual adult figure of 7,500 is reached. Now, what I wish to emphasize is that, with the exception of a transient polynuclear leucocytosis which sets in just after birth, the excess of white cells throughout infancy is due to a **large absolute number of lymphocytes.** You will remember that these cells are derived from the adenoid tissue scattered throughout the body, such as the thymus, lymph glands, Peyer's patches, and Malpighian corpuscles of the spleen, and their excess in the blood indicates a high degree of activity of the adenoid tissue throughout the period of infancy. What the meaning of this activity of the adenoid tissue is it is difficult to be quite sure, but it is probably associated in some way with growth and nutrition; and anything which interferes with the nutrition of the child seems to lead to a still greater degree of activity of this tissue, and consequently to an even more pronounced lymphocytosis in the blood.

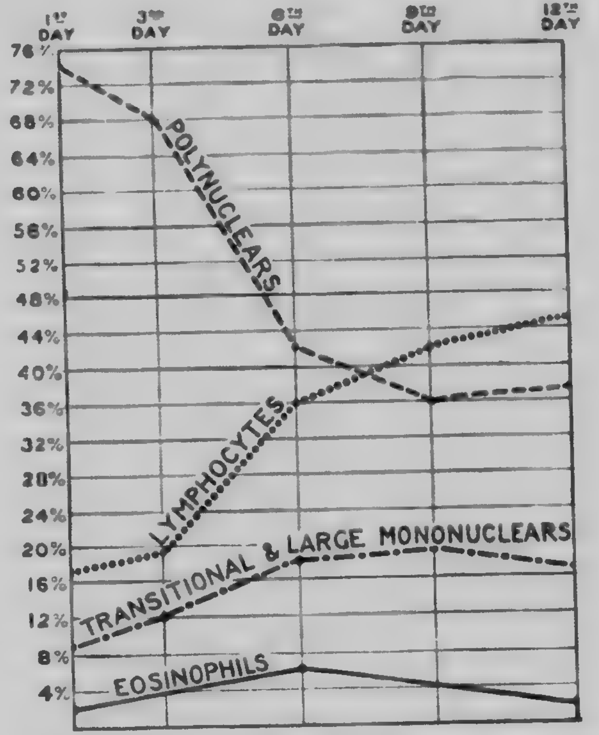


DIAGRAM 13.—DIFFERENTIAL PERCENTAGE COUNTS DURING THE FIRST FORTNIGHT. (AFTER CARSTANJEN.)

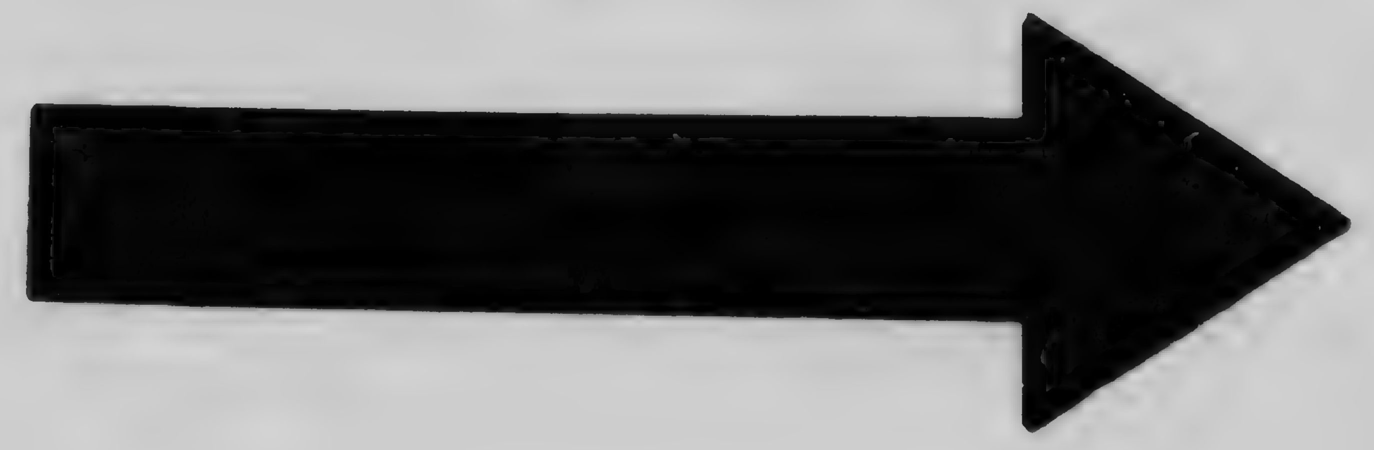
To sum up, then, the **leading characteristics of the blood in infancy** are its poverty in hæmoglobin and the large number of lymphocytes which it contains, and these two facts must always be taken into consideration when you are trying to interpret the meaning of any 'blood picture' in anæmic conditions of childhood.

VARIETIES OF ANÆMIA.

After these preliminary remarks I may pass on to consider the clinical varieties of anæmia met with in children. At the outset we are met with the **difficulty of classification.** It is very probable—indeed, almost certain—that anæmia is always symptomatic—*i.e.*, is the result of some cause which produces increased destruction of blood or interferes with its formation—but we do not yet know what these causes are in every case, and so, for convenience of description, I propose to deal with the blood disorders of early life under the following heads: (1) Congenital abnormalities of the blood; (2) primary anæmias—*e.g.*, pernicious anæmia and chlorosis; (3) secondary anæmias; (4) anæmia associated with enlarged spleen; (5) leukæmias; (6) affections of the adenoid tissue.

1. Congenital Abnormalities of the Blood.

Every now and then you will meet with children who are said to have been pale since birth, and on examination you discover nothing but a more or less marked general anæmia. I think you will find that in a considerable proportion of these cases the child is a twin, and I wish to lay some stress upon the tendency of twins to suffer from anæmia. It would seem as if the mother was sometimes unable to supply enough iron to provide for the proper formation of blood in both twins, and one or other has to go short, with the consequence that anæmia results. The administration of iron by the mouth may speedily improve matters in such a case, but sometimes it fails, and the condition may even prove fatal. When that happens I am inclined to think that there may actually be a congenital defect of the marrow present, which



MICROCOPY RESOLUTION TEST CHART

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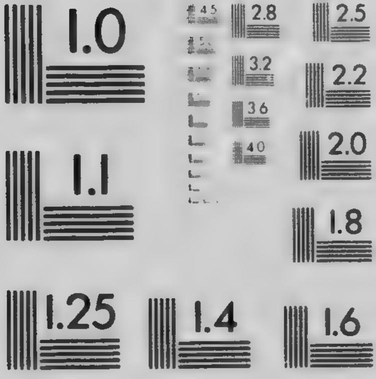

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renders it incapable of forming blood in sufficient amount. Exactly the opposite state of things to this is found in **congenital cyanosis** such as occurs most conspicuously in congenital heart lesions. Some of you may have seen a baby which was shown lately at our Medical Society, and which was the subject of congenital pulmonary stenosis with extreme cyanosis. The red cells in that case numbered no less than 8,000,000 per cubic millimetre, and the hæmoglobin amounted to 110 per cent. The reason for the excess of red cells in these cases has not been satisfactorily explained, though there are many hypotheses about it, but it may, perhaps, be an attempt to meet the difficulty of oxidation. Post-mortem examination in such cases shows that the blood-forming organs are all in a state of extreme activity, and that the fœtal method of intracapillary red-cell formation persists to a large extent.

2. THE PRIMARY ANÆMIAS.

In adults the two chief forms of so-called 'primary' anæmia are chlorosis and **pernicious anæmia.** Of the latter I would only say that it is for some reason or other extremely rare in childhood, and is practically never met with at all during infancy. That is a fact to bear in mind in diagnosis. **Chlorotic anæmias,** on the other hand, are not uncommonly seen in early life. The best examples of them I have seen have been in rickety children who have been fed on a diet containing an insufficient quantity of protein and iron, both of which are so essential for healthy blood-formation, or in older children who have not been supplied with a sufficient amount of animal food. Here is an example: A girl of three years had been fed on the breast till she was eighteen months old; since then she had been rickety. She had a chlorotic complexion, a palpable spleen, and loud hæmic murmurs. The red cells numbered 5,000,000, the white 13,000, whilst the hæmoglobin was only 31 per cent. Under full diet and iron in large doses the hæmoglobin rose in six weeks to 62 per cent., which is little short of the normal at this time of life.

That too prolonged nursing leads almost inevitably to anæmia I have no doubt at all, and the explanation of the fact is probably to be found in the poverty of iron in human milk. You must never forget that the infant does not depend by any means entirely upon the iron in his diet for the supply of that metal necessary to the formation of blood. A considerable proportion of it is derived from the storehouse of iron in the liver with which every child enters the world. About the end of the natural period of nursing, however, this store gets exhausted, and if the diet is then poor in salts of iron anæmia results.

3. THE SECONDARY ANÆMIAS.

I have already said that it is probable that some day we shall regard all anæmias as being really secondary in origin ; meanwhile, however, it is well to reserve that term for those cases in which poverty of blood comes on as a direct consequence of some illness or debilitating condition. Now, it would appear that the blood of the infant is peculiarly 'vulnerable'—that is to say, it undergoes destruction more readily than that of the adult, and so you will not be surprised to hear that secondary anæmia is a very common occurrence in early life. The production of secondary anæmia at this period is, no doubt, aided by the small reserve power of blood-formation which the infant possesses, owing to the complete filling of the bone marrow by red cell-forming tissue even under normal conditions. To this, however, I have already referred.

It is hardly necessary to point out that the possible **causes of secondary anæmia** in childhood are exceedingly numerous ; any condition adverse to the general health of the child might, indeed, be fairly brought within the limits of such a category. For practical purposes, however, I would ask you to bear in mind that the most potent causes of secondary anæmia in childhood are these : (1) Chronic gastro-intestinal disturbance ; (2) the toxins of acute infective diseases ; (3) 'cachexias' of various sorts, notably congenital syphilis and tuberculosis.

That anæmia is apt to result from **chronic catarrh of the stomach and bowels** is well known. There are probably several causes at work in its production in such a case. Diarrhœa leads to deficient absorption and an inadequate supply of the materials from which the blood is built up. In addition to this it is probable that toxic substances are absorbed from the alimentary canal in such a case, possibly the products of the growth of an abnormal intestinal flora, which directly lead to blood destruction.

Of the **infective fevers** as causes of anæmia two stand out with great prominence ; one is diphtheria, the other acute rheumatism. The latter, indeed, leads to such rapid destruction of blood that one would almost be justified in speaking of a 'rheumatic anæmia.' It is especially, I think, when rheumatism lays hold of the heart that severe anæmia is apt to supervene. When going round the wards, for instance, one is often struck by the sudden supervention of marked pallor in a child with acute rheumatism, who has hitherto been going on well, and on examining the heart in such a case one will often find that endocarditis or pericarditis has made its appearance. The moral of this is that you should always remember to put your rheumatic cases through a thorough course of iron during their convalescence.

Of the **cachectic conditions** which tend to lead to secondary anæmia I would specially mention congenital syphilis, in which blood destruction may attain a high degree. Mercury in such a case may actually produce the effects of a hæmatinic. Tuberculosis, too, is a potent destroyer of the blood, especially, I think, when it affects the alimentary canal, and I have often seen cases of severe and unexplained anæmia in which extensive tuberculous ulceration of the bowel was found at the necropsy.

No matter how a secondary anæmia is produced, the **changes in the blood** are the same, and depend more upon the potency of the destructive influence than upon its nature. The hæmoglobin always seems to suffer first, and so in the mildest degrees one finds merely a chlorotic type of blood. In the severer forms the red cells are also reduced in number, and may show some inequality of size and irregularity in shape (poikilocytosis), whilst in the most severe degrees of all, and especially if the general nutrition suffers, an increased number of lymphocytes appears in the blood-stream (lymphocytosis).

4. ANÆMIAS ASSOCIATED WITH ENLARGEMENT OF THE SPLEEN.

From time to time cases will be brought to you of which the following may serve as a fairly typical example :

The child is somewhere between one and two years old. At the first glance you are struck by the creamy tint of the skin and the pallor of the mucous membranes. Proceeding to your physical examination, you make out the signs of more or less pronounced rickets, and on passing your hand over the head you will probably be struck by the presence of frontal and parietal bosses (the so-called hot-cross-bun head). On proceeding to palpate the abdomen you come at once upon an enlarged spleen, which may extend down to or even somewhat beyond the umbilicus. The liver also you find to be notably enlarged, and the superficial lymph nodes may be palpable (see Frontispiece).

On auscultation of the heart you detect hæmic murmurs, and there may perhaps be some moist sounds at the bases of the lungs. When you come to examine the blood you find that it exhibits great poverty in hæmoglobin, along with a more or less pronounced diminution in the number of red cells. The white cells, on the other hand, are increased, often very markedly so, the increase affecting chiefly the lymphocytes. Stained films show that the red cells are unequal in size, and more or less irregular in shape, and that a considerable number of them are nucleated, whilst on examining the white cells more minutely you are struck by the fact that 'large lymphocytes' are numerous, and that you can often see a considerable number of typical myelocytes.

Such cases as this you will find described in the text-books by different names. Some people speak of them as cases of **'splenic anæmia of infancy,'** others as **'pseudo-leukæmia infantum** of Von Jaksch. I propose to adopt the former term, for the word 'pseudo-leukæmia' is one fraught with much possibility of confusion, but I would only remind you that you must not suppose that they have anything at all to do with the so-called splenic anæmia which you are familiar with in the case of adults. On the contrary, the two diseases are totally distinct. There has been much discussion as to the true **nature of this disease,** but there would be no use in my attempting to enumerate the different theories which have been held in regard to it. You need only know that there are at present two chief ways of looking at it. According to one school it is merely a secondary anæmia, whilst according to the other it is a special form of anæmia peculiar to infancy—a disease, in short, *sui generis*. As to the primary disease to which splenic anæmia is supposed to be secondary there is no universal agreement. Some have attributed it to rickets, others to congenital syphilis. That the former condition is almost invariably present all observers are agreed, but to regard it as the *cause* of splenic anæmia is a totally different affair. All the evidence, indeed, points rather in the direction of both the rickets and the anæmia being the result of some common cause. The view that congenital syphilis is the causal factor is at once disposed of by the fact that in fully half the cases no evidence of such disease can be found. The opinion that the splenic anæmia of infancy is a disease *sui generis* has much to commend it, but hitherto no real light has been thrown upon its etiological factors. Some have attributed it to the absorption of a toxin of intestinal origin which exercises a destructive effect upon the blood, but against this is the fact that in quite a large number of the cases there is no evidence of any gastro-intestinal disorder. There, I fear, I must leave the matter, only saying that my own view is that although this disease is almost undoubtedly secondary in the sense of being the result of the action of some destructive influence on the blood, yet we are still quite in the dark as to what this destructive influence may be. It is probably, however, a specific agent of some sort, and in so far the disease may fairly be regarded as one *sui generis*.

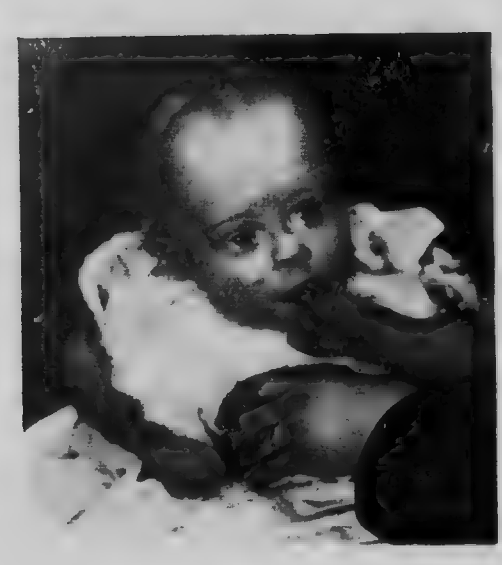

FIG. 63.
'HOT-CROSS-BUN' HEAD.

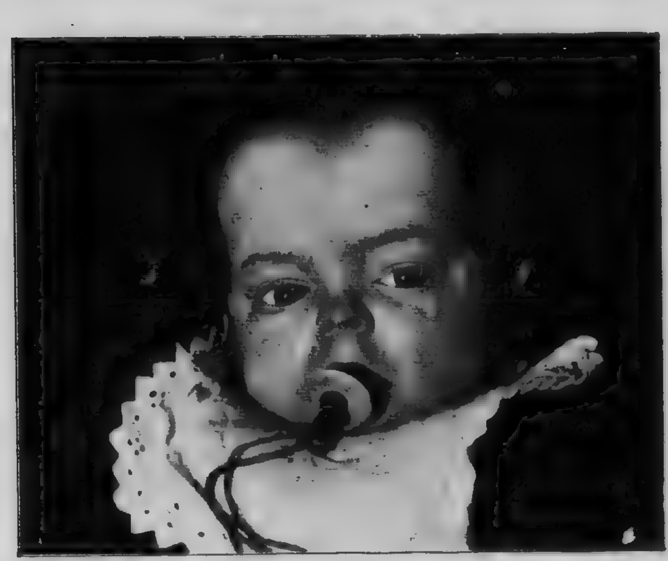

FIG. 64.
HEAD BOSSING.

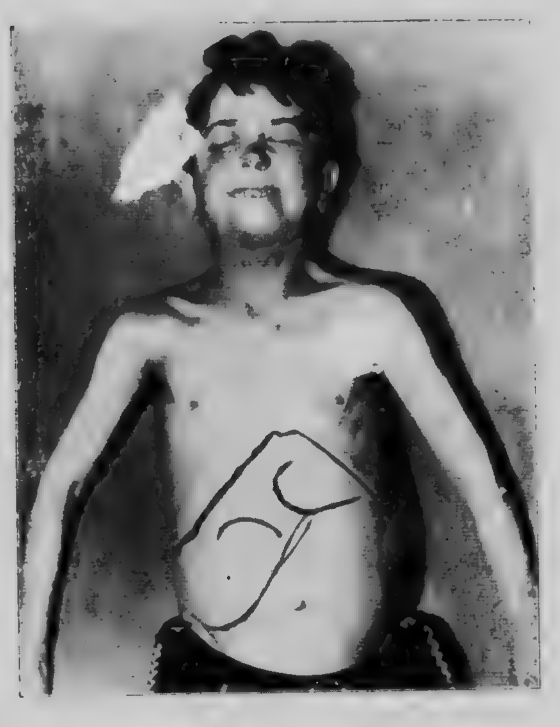

FIG. 65.

ENLARGEMENT OF LIVER AND SPLEEN, THE RESULT OF CONGENITAL SYPHILIS.

Note also the notched incisors.

You will have no difficulty in the **diagnosis** of this form of anæmia. Your troubles will arise when you come to the question of prognosis. Here I should advise you to be guided by the characters of the blood. The greater the degree of leucocytosis and, in particular, the larger the number of myelocytes present, the more grave must the case be regarded. Remember, however, that quite a number recover, though the duration is always one of months, and many exhibit for years a large spleen, which is left behind, high and dry, as it were, after the anæmia has quite disappeared. What you have most to dread is the supervention of intercurrent disease, particularly of bronchopneumonia, by which, indeed, the fatal result, if it ensues, is usually brought about. In older children—usually about or some time after the second dentition—you will sometimes meet with great enlargement of the spleen, with or without some degree of anæmia. A *few* of these cases may be examples of **splenic anæmia of the adult type,** but a greater number are the result of **congenital syphilis,** and you should therefore always make a point of inquiring carefully into the history of such cases, and of looking for other signs of inherited specific disease. Hodgkin's disease may also be attended by great splenic enlargement, but the coexistence of glandular swellings should keep you right.

Of **other causes of splenic enlargement** in childhood, such as Bovaird's 'spleno-megalie primitive,' lymphosarcoma of the spleen, primary splenic tuberculosis, and the enlargements associated with waxy disease and ulcerative endocarditis, I do not propose to speak, for they either belong to the extreme rarities of medicine, or are dealt with in the ordinary medical text-books.

5. Leukæmia in Childhood.

There are, as you know, two chief forms of leukæmia: (1) the *myeloid*, in which cells of the bone-marrow type enter the blood-stream ; (2) the *lymphatic*, in which the blood gets flooded with an excess of lymphocytes. Now the former of these is, like pernicious anæmia, almost unknown in early childhood in the form in which it is usually met with in the adult.* Why this should be I do not know, but if you search through the literature of the subject you will scarcely find a single case of genuine chronic **myeloid leukæmia** in childhood on record. It will be well to bear this simple fact in mind if ever you feel inclined to make a diagnosis of myeloid leukæmia in a child.

Lymphatic leukæmia, on the other hand, is by no means very uncommon in quite early life, though it is probable that it is still often overlooked. Cases will present themselves to you in three chief forms :

(1) Those which exhibit profound anæmia with general glandular enlargement and a hæmorrhagic tendency in the later stages.

(2) Those in which the tendency to hæmorrhage is exhibited from the outset, so that the case resembles one of infective purpura.

(3) Pseudo-scorbutic cases, in which lesions in the buccal cavity (spongy gums, ulcerative stomatitis, etc.) are the most striking feature.

The spleen is enlarged in most, but by no means in all, of these cases, and it rarely attains the size that it does in splenic anæmia. The glands also are not invariably enlarged, and are often little more than easily palpable. The mistake you are most likely to make is in regarding such cases as examples of purpura on the one hand, or of scurvy on the other. I remember going round the wards here on one occasion and finding a child, recently admitted, who was suffering from profuse bleeding from the gums and fœtid ulceration of the buccal cavity generally. By the bedside stood a bottle of lime-juice, and I was told that the child was suffering from scurvy. On examining a blood-film, however, there was found to be an enormous increase of the lymphocytes, which at once made it clear that the case was one of lymphatic leukæmia. In any doubtful case, then, examine the blood at once, and you will avoid error. There is only one thing I would warn you of, and

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* *Acute* myeloid leukæmia is probably commoner in childhood than has hitherto been supposed, but clinically it is identical in its symptoms and course with acute lymphatic leukæmia, and can only be distinguished by a careful examination of stained films, which show the characteristic myeloblasts in large numbers.

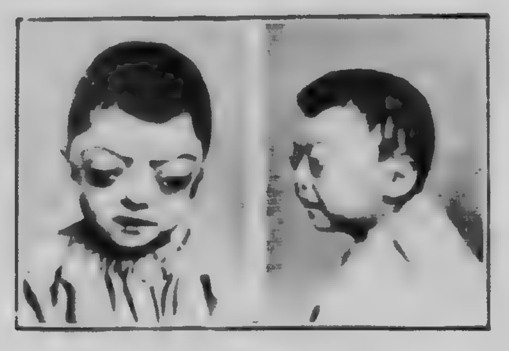

FIG. 66.

CHLOROMA, SHOWING PROTRUSION OF EYES.

(By permission of Dr. Melville Dunlop.)

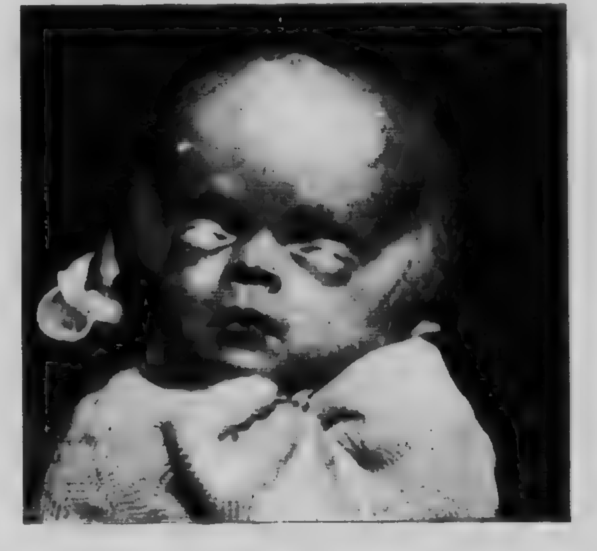

FIG. 67.

SARCOMA OF SKULL: PERIOSTEAL GROWTHS AND BULGING OF EYES SIMULATING CHLOROMA.

that is that you must not expect to find that there is invariably an *absolute* increase in the number of white corpuscles in these cases. What you will always find, however, is that the lymphocytes are relatively greatly in excess—usually numbering upwards of 90 per cent. of the total white cells present.

Closely allied to lymphatic leukæmia is the disease termed **chloroma.** This is a rare condition, of which you may never meet with an example, although it does occasionally occur, even in quite little children. It has the same blood-picture as lymphatic leukæmia, but differs from it in that local tumours develop, often in the periosteum of the [illegible] of the skull and about the orbit, so that the eyes may bulge out. These tumours have on section a peculiar green colour, the exact cause of which is still disputed, and from which the disease derives its name. If you remember the existence of the disease and take care to examine the blood, you are not likely to miss it if ever a case should chance to come under your observation. Ordinary secondary sarcomatous deposits may produce similar tumours about the head and orbits, and I have also known hæmorrhage into the orbit from scurvy simulate it pretty closely, but neither of these exhibit the great degree of lymphocytosis which is characteristic of chloroma.

6. AFFECTIONS OF ADENOID TISSUE.

The great extent and activity of the adenoid tissue in early childhood seem to render it peculiarly liable to become the seat of pathological processes. Of the local collections of this tissue the **thymus** is by far the most important. I cannot deal fully with the diseases of the thymus, nor are they, indeed, of much clinical interest, but I wish to speak briefly of its hypertrophy. We have, unfortunately, no very accurate criterion of what the normal weight of the thymus should be. All we know is that it attains its maximum weight relative to that of the body as a whole at birth, after which it slowly declines till about the second or third year, when it begins to atrophy with increasing rapidity. If, however, you take the trouble to weigh the thymus in many infants of the same age you will find that it varies very greatly, and without, apparently, any special reason. Such, at all events, has been our experience here, and the same thing has been found by all those who have given attention to the subject. Notwithstanding this normal variability, you will meet every now and then with cases in which the thymus is obviously hypertrophied, and sometimes very markedly so. I cannot promise that you will be able to recognize such cases clinically, for the only sign they exhibit is an increased dulness behind the manubrium of the sternum, and that may be produced by other causes, such as enlarged bronchial glands. More commonly such cases are only recognized on the post-mortem table, and you will find that almost invariably they have been cases in which death has occurred suddenly and without apparent cause, and often in otherwise very healthy-looking infants. To such cases the term **status thymicus** or **status lymphaticus** is applied, or they are spoken of as instances of 'thymic' death. Great mystery attaches to the cause of the fatal result in these children, and many theories have been propounded to account for it. According to one set of writers the enlarged thymus acts mechanically, by direct pressure upon the trachea or upon the nerves in the chest. Others hold that the hypertrophy of the thymus in such cases is merely a part of a general overgrowth of lymphoid tissue, which is accompanied by a hypoplasia of the large vessels, which results in a general feebleness of resistance, so that death may ensue upon quite trivial provocation. Which view is right I am quite unable to tell you, but you should bear the condition in mind, for it is sometimes of medico-legal importance. Some cases of supposed 'overlying,' for instance, may really be due to this cause.

Of **diseases** specially **affecting the glands** in early life I would mention infective processes, such as tuberculosis, Hodgkin's disease (which is probably also a chronic infection), and malignant processes such as lymphosarcoma. Of these I do not need to speak in detail, for they are dealt with in every text-book of medicine. This only I would say: that the distinction between **Hodgkin's disease** and chronic multiple glandular tuberculosis is, clinically speaking, a matter of the greatest difficulty, and often, I believe, an impossibility. Nor is the distinction one of much practical importance, for the course and result of the two processes is identical. The blood is the only diagnostic criterion of any value. A polynuclear leucocytosis is, in the absence of any intercurrent pyrexial disease, in favour of a diagnosis of tuberculosis, whereas a relative increase in the lymphocytes speaks rather for Hodgkin. But this distinction is not invariably applicable.

TREATMENT OF THE ANÆMIAS OF INFANCY.

I have left to the last the consideration of the treatment of the anæmias of infancy because the same principles are more or less applicable in all varieties. The first thing to be done is to ascertain the cause if you can, and, having done so, to remove it. If, for instance, the anæmia be dependent upon a syphilitic cachexia, you must administer mercury; if it be due to chronic gastro-intestinal disturbance, that must be remedied by appropriate feeding and medicines. In all cases a due supply of fresh air, and particularly of sunlight, is a great aid in restoring the blood to a proper condition. You will often require to increase the amount of iron-containing constituents in the **diet.** Yolk of egg is one of the best foods for this purpose, for not only does it contain organic compounds of iron, but the lecithin which is so abundantly present in it is also a constituent of the red blood corpuscles. Oat flour and raw-meat juice are also important iron-containing foods. These measures are of greater value in treatment than are **drugs,** but the latter can, of course, give you great help. The milder preparations of iron—of which there are so many available now—are the most suitable for children, and cod-liver oil may require to be given at the same time. I have found the combination of these in the form of the preparation known as 'Ferroleum' very useful. I cannot recommend arsenic very strongly in the ordinary anæmias of childhood, though it seems to be the only preparation we possess which has any influence over Hodgkin's disease. The ordinary form of leukæmia in childhood (the lymphatic) does not appear to be affected by it. For the splenic anæmia of infancy there is no specific treatment—it must be managed on the lines already laid down, special attention being paid to proper feeding. I have tried various preparations of bone-marrow in it, but the results have not been very satisfactory. I should add that in some of the more intractable forms of anæmia in childhood you will get no benefit from iron until you combine it with an aperient, just as is the case in some of the anæmias of adults.

THE PURPURAS.

I wish, before concluding this lecture, to say a few words about purpura, which is commoner in children than in grown-up people. It is not, however, a disease of quite young children, the majority of cases occurring in the second ten years of life.

The essential lesion of purpura is hæmorrhage into the skin, and you will recognize purpuric spots by the fact that they do not fade on pressure. You will be apt to mistake them for flea-bites, but there is usually a darker central spot in a bite which will enable you to recognize it. Sometimes, however, when the bites are fading, the distinction is by no means easy.

Several varieties of purpura are described, and you must distinguish, in the first place, between *secondary purpura* on the one hand and *primary purpura* on the other.

In **secondary purpura** the hæmorrhagic eruption occurs in the course of some other disease, such as one of the acute specific fevers, or in any cachectic condition. It is only of prognostic value, and indicates a severe toxæmia or advanced debility, which often precedes a fatal termination of the illness.

Several varieties of **primary purpura** are described, but they are probably simply different manifestations of one pathological state, although what that underlying state is we do not in the least know, and I do not propose to take up any time with a discussion of the various views which are held about it. For convenience, we may divide the clinical forms of primary purpura into the following groups :

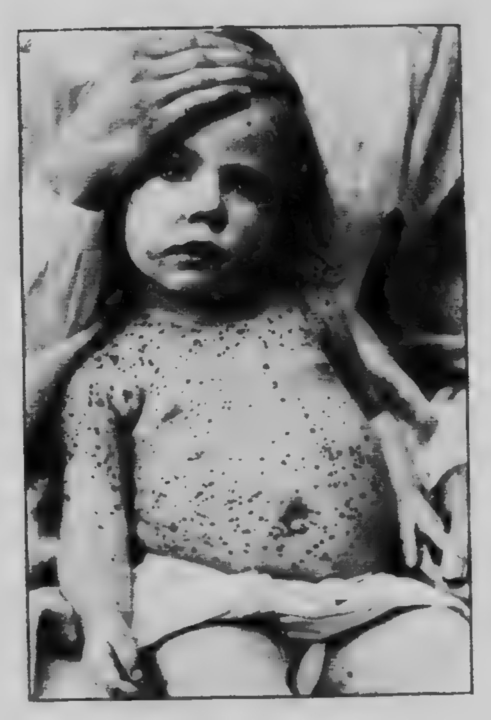

Fig. 68.

PURPURA SIMPLEX.

1. *Purpura simplex*, in which hæmorrhage takes place into the skin only. The photograph (Fig. 68) shows the distribution of the eruption in a typical case.

2. *Purpura hæmorrhagica*, which is a more severe form of simple purpura, hæmorrhage taking place from the mucous membranes as well as into the skin.

3. *Purpura rheumatica*, also known as *peliosis rheumatica*, but better called 'arthritic' purpura, in which joint pains occur as well as hæmorrhages. It has probably nothing whatever to do with rheumatism.

4. *Purpura abdominalis*, or Henoch's purpura, in which attacks of abdominal pain, sometimes attended by vomiting and diarrhœa, are a prominent symptom. The colicky pain is probably caused by exudation taking place into the wall of the bowel, and it is of importance, because it may be so severe as to lead to a diagnosis of acute abdominal disease, such as intussusception. I have known at least one case in which laparotomy was performed on account of it. The moral is that you should always examine the skin very carefully for purpuric spots when you are called to a child who has been attacked by sudden and violent colic.

In the **diagnosis** of purpura you must first make sure that you are dealing with a real purpuric eruption. I have already stated that the characteristic point here is that the eruption does not fade on pressure. Use a microscope slide or a glass lens to make sure. All *erythematous* eruptions fade on pressure. I have already told you how to distinguish bites.

Assuming that you are satisfied that the eruption is purpuric, the next thing you have to settle is whether you are dealing with the disease purpura, or merely with a purpuric eruption as a symptom. The other diseases which you are most likely to mistake for purpura are *acute leukæmia, scurvy, and hæmophilia*. You will distinguish the first of these by examining the blood; the second by inquiring into the diet, and, if necessary by the therapeutic test of giving antiscorbutics; the third by the family history.

The **prognosis** of purpura is good, but you must be prepared for relapses, which are the rule rather than the exception. Some children, indeed, continue to have attacks every few months until after the age of puberty, when they usually cease. Always look out for nephritis in the course of purpura, especially in the abdominal type. It may materially affect the chances of recovery.

Treatment is not very satisfactory, seeing that we know nothing of the cause of the disease. The one essential is *rest in bed*. If you attend to that, it does not matter much what else you do or leave undone. I have tried most of the remedies recommended to lessen the hæmorrhagic tendency, but cannot say that I have had much success with any of them. Salts of calcium I have found no benefit from, although perhaps you ought always to try the lactate, but in some cases oil of turpentine has certainly appeared to do good. Arsenic is recommended by many. Aromatic sulphuric acid is worth trying if there be hæmorrhages from the mucous membranes. Epistaxis and bleeding from the gums you will treat on general principles by the use of adrenalin and other styptics.

In severe cases of purpura hæmorrhagica antistreptococcic serum has been strongly recommended, 10 c.c. being given by the rectum, and repeated as is found necessary ; but you are not likely to need to have recourse to it often.

Seeing that an attack of purpura leaves the child anæmic, iron should be given during convalescence.

LECTURE XXVI

SOME COMMON AFFECTIONS OF THE GENITO-URINARY SYSTEM IN CHILDHOOD

GENTLEMEN,—In the present lecture I propose to deal with some of the commoner affections of the genito-urinary system in children, but shall not refer to such features of these affections as are common both to children and adults.

It is usual when describing diseases of the urinary system in childhood to begin by pointing out some respects in which the urine in childhood differs from that of adult life. Such differences may be summed up for clinical purposes in the statement that the child's urine is paler, of lower gravity, and relatively less acid than that of the grown-up person. It would be of some importance, however, to be able to tell you the amount of urine secreted daily at different ages, but I find that to be impossible, for if you refer to the figures given by different authorities on the subject you will discover such discrepancies as to make their estimates of little value for clinical purposes. Perhaps the estimate arrived at by Dr. Still is as good as any other, and has the advantage of being easily remembered—viz., that after the age of four the age of the child in years multiplied by two and a half gives the average amount of urine in ounces. Below the age of four, however, the secretion is relatively greater than this, because of the more fluid character of the diet.

I have occasionally had children sent to me because they were supposed to be passing too little urine, but I have always found on investigating such cases that the amount secreted was not really less than the minimum average for that age. Every now and then, however, you will meet with cases in which no urine is passed at all for some hours, and in which the bladder also remains empty. These are cases of true suppression, or **anuria.** The cause of this condition is quite obscure, but it need occasion you no alarm, for it never lasts longer than twenty-four hours, and usually a hot bath and a dose or two of spiritus etheris nitrosi is sufficient to restart the secretion. The opposite condition —**polyuria**—may also occur without apparent reason for brief periods, but when continued should make you think of diabetes mellitus or insipidus.

Not unfrequently children pass uric acid **gravel.** I have told you in a previous lecture that the pain caused by this may closely simulate that of intestinal colic, but that the condition can usually be recognized by the detection of grains of uric acid sand on the napkins. The indication in these cases is usually to reduce the amount of food, and always to administer citrate of potash liberally.

At other times a condition is met with which one can only describe as '*uric acid storms.*' These may occur in infancy, but are perhaps commoner in children about three to four years old. They often take place at very regular intervals, exhibiting a decided periodicity. The occurrence of the 'storm' is heralded by a day or two during which the child is pale, languid, and generally 'out of sorts.' There may also be an ethereal odour in the breath, and the stools may be pale. These symptoms are followed by an attack of violent screaming, and some hours later gravel is passed, or it may be noticed as a deposit in the urine without there having been any signs of pain.

The pathology of these cases is obscure, but I strongly suspect that they are in some way due to a functional disorder of the liver, and are closely allied to other periodic affections, such as migraine, 'food fever,' cyclical vomiting, and so-called 'bilious attacks.' Like these disorders, too, they are most successfully treated by cutting down the carbohydrates in the food—*not* by reducing the proteins, *e.g.*, meat, as is often done—and by the regular use of an 'hepatic' aperient, such as rhubarb and gray powder. In addition, of course, citrate of potash should be given freely during the attack.

Another disorder which may be due to the presence of uric acid crystals in the urine, or to excessive urinary acidity, is what is called **vesical spasm.** It may closely simulate vesical calculus. I remember a medical friend of mine in the country ringing me up one morning on the telephone to ask my advice about a surgeon to whom to send one of his children, because he believed the child to be suffering from stone. On asking the grounds for this opinion, I was told that there was at times great pain and strangury on micturition. I advised that before a surgeon was consulted the child should be given a course of citrate of potash and hyoscyamus. This was done, with the result that the symptoms quickly disappeared. That was a case of vesical spasm. Of course, **calculus** is not very uncommon in children, too, but as its symptoms and the methods of diagnosing it are the same as in the case of adults, I need not say anything more about it, except to point out that sometimes the chief symptom is incontinence, and that when a child suddenly develops enuresis calculus is always one of the things to think of. I recall, for instance, the case of a little girl whom I saw many years ago, who was brought to the hospital for incontinence along with some symptoms of cystitis. On sounding the bladder we found a large stone, which we were able to extract by dilating the urethra, with immediate relief to all her symptoms.

INFECTION OF THE URINARY TRACT.

I wish now to turn to a totally different subject—namely, *infections of the urinary tract in childhood*, by which one means for practical purposes infection by the *Bacillus coli*. Now when the *B. coli* finds access to the urinary tract, one of four conditions may result : (1) A simple bacilluria ; (2) cystitis ; (3) pyelitis ; (4) suppurative nephritis. Or these may occur in various combinations.

(1) *Bacilluria* alone gives rise to few symptoms. There may be some disturbance of the general health, but nothing very definite, and the condition can only be diagnosed by an examination of the urine, which is opalescent and does not clear on filtering. It usually remains acid—for the *B. coli*, as you know, does not split up urea—but has often a

peculiar fishy odour. Microscopical examination shows it to be swarming with bacteria, which on culture prove to be the *Bacillus coli*.

(2) The symptoms of *cystitis* due to *B. coli* I need not describe to you in detail, as they are the same as those of any other cystitis except that the urine remains acid unless a secondary infection takes place.

(3) *B. coli pyelitis* is met with in its most striking form in young infants, most frequently, so far as my experience goes, in girls. The constitutional symptoms are much more pronounced than the local, and may quite overshadow the latter. They consist in high irregular fever, the temperature often running up and down in the wildest manner, and often there are rigors. In spite of the fever the general health may remain surprisingly good, but there may be some signs of collapse when a rigor occurs. Such a case may be difficult of diagnosis if your mind is not open to the possibility of pyelitis, but if you only think of that possibility there is usually no difficulty, and on inquiry you will generally find that there is some frequency of micturition, probably accompanied by more or less pain, and that the water has been noticed to be thick. Bacteriological examination will set all doubts at rest. The moral is that in all cases of fever of doubtful origin in childhood one should examine the urine for *B. coli*.

(4) *Suppurative nephritis* is fortunately a rare result of an infection, and is usually, perhaps always, fatal. It is distinguished by the ordinary signs of an acute nephritis, and is usually associated with evidences of pyelitis or cystitis as well.

The **prognosis** in cases of infection by the *B. coli* is on the whole favourable, but cases are often very obstinate to treat. It is not usually difficult to get rid of the constitutional symptoms, but to banish the bacillus from the urine altogether is by no means an easy matter, and it is apt to persist off and on for long periods. Death also sometimes results. Thus Dr. John Thomson found that out of twenty-four cases of *B. coli* pyelitis in infancy three died, and out of sixty cases at all ages treated recently at Great Ormond Street Hospital six proved fatal (Jeffreys).

You may now ask, *How does the B. coli get into the urinary tract at all?* This question has not been definitely settled, but there are three possibilities. (1) Infection might be by the blood-stream, the bacilli being excreted by the kidney. There is reason to believe that this rarely occurs. (2) The infection might be from below upwards, the bacilli getting up the urethra. This view has been widely held, and the alleged greater frequency of cases in girls has been believed to support it, the idea being that the short female urethra is more easily traversed. (3) Infection might be direct from the bowel to the kidney. Investigations made at the Children's Hospital tend to show that this is the usual route of infection, the bacilli passing direct from the ascending colon to the right kidney, the transference being perhaps favoured by some damage to the mucous membrane of the bowel.

In some cases, at least, a diseased appendix which was adherent to the kidney has afforded the means of infection.

Seeing that the bowel is the primary source of the organisms in all cases of *Bacillus coli* infection of the urinary tract in children, one obvious part of **treatment** must always be to disinfect the colon as far as possible. This can be done by the use of large enemata, and I have faith also in the use of calomel in doses of $\frac{1}{10}$ grain two or three times a day, although I do not believe much in other so-called intestinal antiseptics. If there be any reason to suspect disease of the appendix, that organ should be removed. The rest of the treatment must depend to some extent upon the special site of the infection. If there be cystitis, washing out of the bladder by the injection of iodoform emulsion is useful. In cases of pyelitis I have found great benefit from the use of citrate of potash in large doses. It is almost a specific in the cases of *coli* pyelitis in infants, but it must be given freely and one must go on increasing the dose until the urine is alkaline. Surprisingly large quantities are sometimes necessary to effect this, 15 grains three or four times a day being none too much in many cases. So soon as the urine is alkaline the temperature and other constitutional signs usually subside in the most dramatic fashion.

In older children the alkaline treatment is not so successful, but is always worth trying, and the use of urotropin may be combined with it.

The same measures are applicable in the cases of pure bacilluria.

In chronic obstinate cases of all forms the use of a vaccine preferably prepared from the urine of the patient, is always worth trying, but I would warn you not to expect too much from it. It is true that the constitutional symptoms are sometimes improved, but it is exceptional to find that the vaccination causes the bacilli to disappear altogether from the urine. They are apt, indeed, to persist, or to keep on coming and going in the most aggravating way, and in that case all you can do is to get the child's general health into as good a state as possible—to which end change of air is often a great help—and then to trust to time.

FUNCTIONAL ALBUMINURIA.

I may now pass on to consider some of the commoner affections of the kidney in childhood, and first of all I may say a word about *functional albuminuria*. This condition also known as physiological, orthostatic, cyclical, postural, or intermittent albuminuria—is, as you know, one which has given rise to much discussion. I do not propose to attempt to deal at all fully with the many interesting points which have been raised with regard to it, but I would only say that there can be no doubt (1) that such a condition really exists, (2) that it passes off without apparently leaving the kidney in any way damaged, and (3) that it is of no serious significance. The condition is not really a disease in itself, but merely a symptom, and is especially met with in children who suffer from that state of ill-health which I have dealt with fully in a previous lecture under the title of 'Mucous Disease.'

The important question, of course, is how to tell this form of albuminuria from that of genuine nephritis. Much has been written on this subject, but the chief points of distinction appear to be (1) that functional albuminuria is not present when the child first gets up in the morning, but only comes on after he has been going about for an hour or two ; (2) that granular casts are never present, although the hyaline variety may be. I have observed, too, that in cases of functional albuminuria the urine usually gives a very definite cloud on the addition of acetic acid in the cold alone, whereas this does not happen in cases of organic albuminuria. This point has not received much attention, but I believe it to be worth noting. It means, I suppose, that the protein present in the 'functional' cases is not all true serum albumin, or globulin, but is in part a mucin or nuclein compound. Many theories have been advanced to explain the cause of the passage of albumin into the urine in these cases, but the most convincing is that which attributes the symptom to alterations in the vascular tension in the kidney when the erect posture is assumed.

The suggestion has been recently put forward that the occurrence of the albuminuria is the result of a lessened coagulability in the blood, and it is said that it can be made to disappear by the administration of calcium lactate. I cannot say, however, that I have found this happen in the few cases in which I have tried it, and I am rather sceptical about this theory. As regards the **treatment** of these cases, I believe the only thing to be done is to attend to the general health on the lines laid down in the lecture on 'Mucous Disease,' to which I have already referred, and to leave the albuminuria to look after itself.

I may now go on to say a few words on the subject of *hæmaturia in childhood*.

HÆMATURIA.

A child may be brought to you by the mother with the statement that he passes blood in the urine. There is here one fallacy which I would warn you against, and it is that mothers are apt to regard as blood what is really not blood at all, but a brick-dust deposit of urates. But assuming you have verified the fact that the child is passing blood, there are but few possible causes of it which you have to consider; indeed, I think there are but four causes of hæmaturia in infancy which it is necessary for you to bear in mind. One is **hæmophilia**, which may be a cause of hæmaturia, especially in boys. Another cause is **scurvy**, which I believe is commoner than one is disposed to think, and is often overlooked because examination of the urine of young infants is apt to be omitted. I remember a child who was under the care of a surgeon in a hospital for the passage of blood, and who was believed to be the subject of stone in the kidney. An operation was performed, but no stone was found. A physician then saw the child, and inquired into the diet; the replies given led him to think that it was a case of scurvy, and on putting the child on to fresh vegetables and fruit the hæmaturia disappeared in a few days. This, then, was a case in which failure to remember that scurvy may be a cause of hæmaturia led to the performance of an entirely unnecessary operation. The third cause of hæmaturia is the **passage of urates and oxalates.** The hæmaturia in such a case is usually, I think, paroxysmal or intermittent, coming on and lasting for a few days and then passing off again. Careful examination will show the presence of crystals of uric acid or oxalic acid, and suitable treatment will cause the blood to disappear. The fourth cause is **sarcoma of the kidney,** and one which it is well to bear in mind. Not long ago I saw a case where this gave rise to a great difficulty. It was that of a young child who passed a trivial amount of blood in the urine, but passed it continually. Oxalates were discovered in the urine, and it was thought that they were the cause of the hæmaturia. But special treatment directed to remedying this did not affect the passage of blood, although the oxalates ceased to appear. By-and-by an abdominal tumour became evident, and operation showed it to be a renal sarcoma. Remember always, then, that continuous small bleedings may be a comparatively early sign of sarcoma of the kidney in a young child.

In older children, in addition to the causes already mentioned, you should think of **Raynaud's disease,** of **purpura,** and of **rheumatism** as possible causes of the appearance of blood or blood-pigment in the urine, and in the two former of these hæmaturia may be present as the only symptom. Quite recently I have seen in consultation two girls, both of whom suffered from rheumatic endocarditis and slight hæmaturia. Whether the renal hæmorrhage in these cases was due to embolism or not I cannot say, but it passed off quickly in each case.

NEPHRITIS.

In childhood one has to deal, for practical purposes, with only two varieties of inflammation of the kidney : (1) acute nephritis, and (2) chronic parenchymatous nephritis. It is true that the chronic interstitial form does occur, but it is so rare as to be almost a curiosity.

Acute nephritis presents two varieties—tubular and glomerular. The recognition of these is of importance, not only pathologically, but clinically as well, for the glomerular form has a much better prognosis than the tubular.

Which form of nephritis is produced depends, apparently, upon the poison at work. Some toxins, notably that of scarlatina, seem chiefly to affect the glomeruli ; others, such as the diphtheritic, the tubules. Scarlatinal nephritis is therefore a less severe affection than most other forms of renal inflammation. In addition to the two 'acute specifics' already mentioned, nephritis may more rarely follow measles, chicken-pox, whooping-cough, or even German measles, but in a large number of the cases met with in practice you will find it impossible to say what the cause has been. Many of these cases are attributed by the mother, rightly or wrongly, to chill, but I believe that in a certain number of them the poison finds access to the body through the throat. At all events there can be no doubt that nephritis may follow upon an acute tonsillitis.

The **symptoms** of acute nephritis are so well known that I need not dwell upon them. Œdema is often the first thing noticed, but here I would warn you that œdema in children does not necessarily mean nephritis. There is a form of œdema sometimes spoken of as *toxic or essential œdema*, met with in young children suffering from chronic diarrhœa, which closely simulates the œdema of nephritis, but in which there is no other evidence of renal disease. Its exact mode of production is unknown, but it is not necessarily of serious omen, and I have often known recovery follow even when it has been present to a marked degree. On the other hand, you must remember that although œdema in children does not always mean nephritis, *nephritis may occur without* *œdema.* This is specially the case in little babies, and for that reason nephritis is apt to be overlooked when it occurs as a complication of some other disease in early life.

The **prognosis of nephritis** in children is much better than in adults, and complete clearing up—or apparent clearing up—of the inflammation is fairly common, especially, as I have already told you, in the glomerular form. One reason why acute nephritis cases do so much better in early life no doubt is that there is rarely, if ever, a chronic nephritis at the back of the condition on which the acute inflammation has supervened—a state of things which, as you know, is not uncommon in adults.

Even when acute **uræmic symptoms** have supervened—usually in the form of convulsions—the prognosis is not necessarily bad, for under prompt treatment by hot packs and transfusion these may pass off and all go well.

In the **treatment** of acute nephritis in children, the most important thing is to *keep the patient as long as possible in bed.* I have often regretted letting a child with nephritis get up too soon, but never having kept it long at rest. The diet at first should consist of milk only, but when the acuter symptoms have passed off farinaceous foods may be added, for one must take care not to starve the patient. Medicines are not of much help, but a mild saline diuretic may be given, and to this, if there be much dropsy, a little digitalis may often be added with advantage. Hot packs are also of great service in keeping up a free action of the skin.

Under this treatment the urine either clears up completely, or the condition becomes chronic and the child is left anæmic and more or less dropsical with a considerable amount of albuminuria. When this happens, one has to deal with *chronic parenchymatous nephritis,* which is a very troublesome condition. Such a case, it is true, may drag on for a long time with more or less albuminuria, but sooner or later it is apt to prove fatal.

The treatment of the disease when it has reached this stage is very unsatisfactory, and I know no class of case which is more disheartening to manage. So long as there is much dropsy the child must be kept in bed, and this may mean months. Diuretics seem to have little effect in removing the dropsy, though digitalis is sometimes helpful, and a *salt-free diet* will often do much. Such a diet is not difficult to construct out of milk, farinaceous foods, and fruit with bread, biscuits or scones baked without salt. When the dropsy has disappeared, one must be careful not to over-diet, and the mere presence of albumin is certainly no bar to the moderate use of even red meats. It is easy to retard recovery by too great zeal in sparing the kidney.

A curious phenomenon which often occurs in these cases is *spontaneous diuresis.* The child may be quite waterlogged and the dropsy have failed to respond to all treatment when suddenly a great flow of urine sets in and all the fluid drains away in two or three days. Only too often, however, it returns, and such spontaneous diuresis may recur several times in the course of the case, and should it happen to set in shortly after a new diuretic mixture has been exhibited the mixture is very apt to get the credit.

For the *anæmia* which is always present in cases of chronic parenchymatous nephritis, iron is useful, and may be given either in the form of liq. ferri acetatis combined with acetate of potash, or in the old combination of sulphate of iron and sulphate of magnesia, if it be desired to act upon the bowels. The use of iron seems not only to lessen anæmia, but to favour the restitution of the kidney epithelium, and is therefore actually curative. When all the symptoms have disappeared but some albumin is still present in the urine, change of air to a mild, dry climate is often helpful.

VULVO-VAGINITIS.

I wish now to say a word or two about chronic *vulvo-vaginitis* in childhood, which is by no means an infrequent affection, especially in patients of the hospital class. There are two forms of it—the simple form in which pyogenic organisms are the cause, and the gonorrhœal form which is due to the gonococcus. The latter appears to be the commoner. The view long held that vulvo-vaginitis belongs to the 'strumous' order of diseases and is a sign of general ill-health is, I believe, erroneous. It appears to be really a purely local affection requiring local treatment. The gonorrhœal variety is horribly contagious, and if introduced into a ward may spread round it like wild-fire in spite of stringent precautions. On the other hand, it is a peculiar fact that it rarely leads to other gonorrhœal complications, such as blepharitis, arthritis or salpingitis.

Treatment of vulvo-vaginitis must be thorough and energetic.

In *simple* cases the parts should be thoroughly irrigated two or three times daily with an antiseptic solution (saturated boric acid or 1 in 5,000 perchloride). The solution should be applied with a syringe or a swab, and gauze soaked in it may be left in contact with the mucous membrane. The surrounding skin should be protected by white precipitate ointment (5 grains to the ounce). In the more severe cases silver solutions (1 to 5 per cent. protargol or argyrol) may be used instead of the above lotions. Suppositories containing 1½ grains of salol in cocoa-butter may be introduced into the vagina two or three times daily. General cleanliness should be strictly observed, and the use of a sulphur bath every other night is often helpful.

In the *gonorrhœal* cases treatment must be even more energetic. As I have already told you, this form of the disease is highly contagious, and the patient should therefore be strictly isolated and the utmost care taken to prevent the carrying of infection by napkins, washing utensils, sheets, etc. All such articles should be thoroughly disinfected before being washed. The local treatment is the same in principle as that employed in simple cases, but stronger antiseptics are required (*e.g.*, perchloride 1 in 2,000, protargol 5 to 10 per cent., argyrol 5 to 25 per cent.). A diaper or pad should be worn to prevent the child carrying infection to the eyes. Quarantine should be continued for some time after all organisms have disappeared from the discharge.

In obstinate cases of either form the use of *vaccines* may prove helpful, prepared for choice from cultures obtained from the discharge. In the gonorrhœal variety this treatment has often given good results, even where a stock vaccine is employed. In the case of a child six months old one may begin with an injection of 50 millions, which is repeated every fifth day, the dose being increased by 10 millions each time. After five injections have been given the interval may be extended to ten days.

LECTURE XXVII

SOME COMMON SYMPTOMS OF DISEASE IN CHILDREN AND THEIR DIAGNOSTIC SIGNIFICANCE

GENTLEMEN,—Those of you who have attended much in the out-patient department must have been struck by the fact that the number of actual complaints for which children are brought to the hospital—that is to say, the things noticed wrong by the mother—are comparatively few in number. And that, after all, is inevitable. A young child is unable to express—even supposing he is able to experience—those multifarious abnormal sensations which make up what we call the symptoms of disease in grown-up people. And it follows from this that your diagnosis must always rest mainly upon the results of your objective examination. Now, I need hardly tell you that therein lies one of the great difficulties of children's practice—you get so little help from your patient. The more, therefore, is it necessary for you to study as carefully as you can such symptoms, few though they be, as the friends of the child have been able to observe. I thought, therefore, that we might profitably spend our time in this lecture if we took up together the study of the commoner symptoms which children present, and tried to arrive at some idea of their value in diagnosis.

WASTING.*

The first symptom that I wish to direct your attention to is one which occurs with very great frequency in the out-patient department. It is that spoken of as *wasting*. With regard to this symptom, I would say, first of all, that it may be due on the one hand to very serious organic disease, or on the other hand, to comparatively trivial disorders of health. And what you have always to do when a child comes before you presenting wasting as one of the chief symptoms, is to eliminate so far as you can the possibility of organic disease before you go further. In the case of young infants there are three **organic diseases** which are specially apt to be accompanied by wasting as their chief symptom. The first of these is *congenital syphilis*, the second is *tuberculosis*, and the third is a *latent empyema*. With regard to the first of those there should be no difficulty; you should easily be able, by inquiring into the family history, and by examining for such symptoms as snuffles, cranio-tabes, rashes, and other manifestations of inherited specific disease, to recognize that it is syphilitic, or exclude that disease as a cause of the wasting. If you are still in doubt, you will often find that by the therapeutic test—that is to say, by the administration of gray powder—you can tell within a very short time whether such a taint is present or not; for syphilis may cause wasting, even though present in only a small degree. With regard to *tuberculosis* in young infants it is very much more difficult to be certain; in fact, I would say that often you cannot be certain of it at all, because tuberculosis may have attained a very widespread degree of development before it produces any pronounced physical signs. Often in such cases you must remain in doubt, and watch the effect of treatment. If under treatment directed to the symptoms the wasting still continues and becomes progressive, then your suspicion that you are dealing with a case of tuberculosis is, of course, greatly strengthened. But as a matter of fact it will sometimes be only on the post-mortem table that the nature of the case will be made clear. There is nothing more difficult than to diagnose accurately between a case of tuberculosis in a young infant and one of so-called simple marasmus. With regard to the third of the conditions I mentioned, *latent empyema*, you should have no difficulty. You cannot miss it if you will examine the child carefully and systematically, particularly, of course, at the bases of the lungs. Not long ago a child was sent here with the history that he had suffered from measles a month or two previously, and that he had been wasting ever since. He had been under treatment outside, but had made no progress towards recovery, rather going from bad to worse. He was sent here under the impression that he was suffering from tuberculosis, and it was thought that nothing further could be done. On examination we found marked dulness at one base, with signs of fluid. On putting in a needle pus was obtained, and after its evacuation the child at once improved, and made an uninterrupted recovery. So there is the chance that even where a child is under skilled observation an empyema may be overlooked, because it may have produced no sign other than that of continual and progressive wasting.

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* This subject was also dealt with in Lecture IX., but as it is approached here from a somewhat different point of view, I have allowed it to stand.

Of the non-organic diseases which may produce wasting as one of their chief signs in young infants, the principal are **disorders of digestion** and unsuitable feeding. As a matter of fact, these are by far the commonest causes of this symptom ; either the child does not get enough nourishment, or he gets it in an unsuitable form, with the natural consequence that in both cases continuous loss of weight is manifested. Hence in the majority of such cases your chief duty will be to inquire into the nature of the feeding and of the food given, and to rectify any errors which you find.

When you come to inquire into the causes of **wasting in older children,** you will find again that there is one great organic disease which is particularly apt to produce that symptom, and I need hardly say I refer to tuberculosis. Some of you saw among my out-patients this morning a child, aged three and a half years, who was brought with a history of wasting ; nothing else had been noticed, except that the child sweated at night. On examination, however, we were able to find without difficulty unequivocal signs of fairly widespread tuberculosis. The commonest functional disease which you are apt to mistake for tuberculosis is that chronic form of dyspepsia which occurs in children at about the period of second dentition, and to which I am so often drawing your attention in the out-patient room.

You know the history—the child is sent up with the statement that he has wasted, that he has a cough, that he has no appetite, and perhaps that he has certain nervous disturbances, such as headache or restlessness at night. Such cases are constantly diagnosed as tuberculosis when they are not so at all. And the mistake, after all, is not an unnatural one, for there are many signs connected with them which in the adult one is accustomed to associate with phthisis; the child has been losing weight, has lost appetite, and has a cough. Those who see little of children and much of adults are apt to conclude that these signs mean phthisis in a child, just as they would rightly make one suspicious of phthisis in a grown-up person. You should know that many such cases are simply examples of chronic gastric catarrh associated with pharyngitis, or enlargement of tonsils, or adenoid growths, and they can be perfectly cured if they are put on a suitable diet and the condition of the throat attended to.

COUGH.

I will next pass to the consideration of another symptom which is particularly common in children, and that is *cough*. Of cough as a symptom in children I would say first that it would be a mistake to suppose that it is always, or indeed usually, the result of respiratory disease. The majority of coughs in children are not due to any abnormal condition of the lungs or bronchi at all. It is all the more necessary that you should be warned of this, because frequently the mother makes the diagnosis herself, and instead of saying the child is suffering from cough she will say he has 'bronchitis,' and so you are apt to be misled. Statistics were collected by Dr. Parkinson* from one of the children's hospitals in London on this subject, and he found that of a large number of consecutive cases of cough, fully 40 per cent. were due, not to disease of the respiratory organs, but to abnormal conditions in the throat, whereas only 31 per cent. were due to disease in the chest. So you see there was actually a clear majority of cases in which the cough was due to the throat. I think there is no more important point to realize about cough in childhood than that. The **abnormal conditions of the throat** which may give rise to cough in children are three in number. You may have simple pharyngitis, or enlargement of the tonsils, usually existing with adenoids, or elongation of the uvula. These three conditions may occur together, as I need hardly say, or you may have combinations of any two or all three of them in any one case. Now, from the character of the cough you can often infer what is likely to be its cause in any particular case which comes before you. For instance, in *simple pharyngitis* it is usually a dry tickling cough, occurring at frequent intervals. In *adenoids* it is rather of a choking character, often accompanied by vomiting, and frequently also by epistaxis. On the other hand, the cough which is associated with an enlarged uvula is characterized by the fact that it comes on when the child lies down, because the uvula falls back into the pharynx and irritates the posterior wall.

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* *Brit. Med. Journ.*, 1899, ii., p. 465.

When you come to consider cough arising from **irritation in the bronchi,** you will often have to face the problem, Is it simple bronchitis, or is it whooping-cough ? Now, that is a question which it is not always easy to answer, although it is one to which the mother expects you to be able to give an exact and definite reply. Although you cannot be certain always about the existence of *whooping-cough,* there are certain things which should make you suspicious of it. And first, the cough of whooping-cough is essentially paroxysmal, it tends to occur especially at night, and it is often followed by vomiting. When you examine the child you find that the cough is out of all proportion to the severity of the physical signs. There may be only a few bronchitic signs in the lungs, although you are told that the cough is one of very great severity. You will frequently find also that there is a sublingual ulcer, which is of real value, because it may occur before the cough has begun to take on the true whooping character. There is another thing which you will notice on examining a child whom you suspect to have whooping-cough, and that is that he looks puffy about the eyes ; the congestion of the head and face which the paroxysm occasions leads to puffiness, which to the experienced eye is often suspicious. You will be apt to mistake for whooping-cough the cough which you get from pressure upon the nerves in the chest caused by **enlarged bronchial glands.** In such a case the history may be of some value, because you know that whooping-cough rarely occurs twice in the same child, and if there is a history of his having had an attack of whooping-cough, and if he develops a similar sort of cough again, the chances are that it is not true pertussis on the second occasion. The other sort of cough which may simulate whooping-cough is that which you get in *bronchiectasis*. But bronchiectasis is not particularly common in children, and from the clubbing of fingers and signs of cavities in the lungs, and the character of the expectoration, there should be no difficulty in making the diagnosis.

When you have excluded the causes of cough which I have mentioned, those in the lungs and these due to the state of the throat, there remain a considerable number in which you can find no definite cause at all. Some of these are certainly due to gastro-intestinal disorders. There is in children, I think, such a thing as **stomach cough ;** at all events, you find children who have a constant cough which persists until the state of the digestive organs is attended to. And some people have even described a cough as the result of the presence of worms and the reflex irritation which they produce.

Finally, there is an extremely uncommon form of cough, but one which it is well to be familiar with, and that is the **hysterical cough.** I remember, shortly after I started seeing out-patients here some years ago, a boy being brought, who, as he sat in the waiting-room, kept on every half minute or oftener giving out a curious kind of short cough. I examined him without finding any cause for it, and from the history of the case and the general impression one got, I came to the conclusion that it was hysterical. Under the influence of suggestion and one or two doses of valerian the cough stopped absolutely in twenty-four hours. More commonly the hysterical cough is not of that sort ; it is of the typical barking character met with in older subjects.

PYREXIA.

The next symptom I wish to consider with you is *pyrexia*, or fever, as an indication of disease in children. I mean by that cases in which pyrexia is the only symptom—where a child is brought to you by the mother because he is hot and feverish, and nothing else has been noticed wrong. These are often cases of great difficulty, and always of very great anxiety in practice, because pyrexia may be, and often is, a sign of serious disease coming on—disease which may be of an infectious nature; and one always wants to make certain at the earliest possible moment what the cause of the fever is. The first thing to look for are signs of the **acute specific fevers.** You should examine the child carefully for one of these, particularly for scarlet fever and measles. In scarlet fever examine the throat for erythema, and look for the early signs of eruption, remembering that it comes out first about the chest and neck. In measles look carefully for catarrhal signs or indications of conjunctivitis and running of the nose. Examine for a rash about the forehead and head. If the rash is not yet out, look for Koplik's spots, which are of very considerable value in the early diagnosis of measles. I need hardly say you should also inquire into the history as regards the possibility of any exposure to infection to one of the acute specific fevers. Failing the discovery of any of those, I advise you to look, especially in young infants, for signs of **pneumonia.** I saw in consultation the other day a baby who had suddenly become feverish about three or four days previously. No physical signs were discovered by careful examination, which had been repeated several times after the child was taken ill. When I saw the child there was little to be made out, but at the tip of one lung there was a small patch of tubular breathing and dulness, indicating the development of apical pneumonia. In addition the child had the characteristic grunting pneumonic breathing; and one had no hesitation in saying that the cause of the fever was a small patch of croupous pneumonia at the top of one lung. That is a frequently overlooked cause of pyrexia, as I know from my own experience. I should therefore advise you in any case of acute fever coming on in a young infant to examine the apices of the lungs with particular care, because a patch of pneumonia so small as to be easily overlooked on physical examination may yet be the cause of very high temperature. Another cause of fever which it is important you should remember is **acute gastritis.** I remember seeing a child a few years ago of about two or three years of age who had been seized with high fever with some abdominal tenderness and vomiting, and the question arose, What was the cause of the symptoms? Various things were suggested, and finally it was thought that the child was suffering from appendicitis, and the possibility of operation was contemplated. But the whole case cleared up entirely after the administration of a few doses of calomel. This was a case of acute gastritis accompanied by high fever. Sometimes, indeed, the fever in such a case is very high, and may be accompanied by rigors. Another disease which you should never forget the possibility of where fever is present and nothing else, is **otitis media.** Usually in such a case there will be signs of earache in addition. The child will manifest pain or discomfort in the ear; and if you suspect inflammation the best thing you can do is to examine the membranes, and if necessary puncture them, and if you do so you may often find a small quantity of sero-purulent matter escape with the immediate relief of all the symptoms.

Another cause of fever in children which you are apt to overlook is **acute rheumatism** where the joints are not affected. Not long ago a boy was admitted to one of my beds here who had acute fever, and in whom careful physical examination revealed absolutely nothing wrong. But there was one suspicious point, which was that, although he had a temperature of 103° F., he was sweating profusely, and, further, he looked like a child of the 'rheumatic diathesis,' as it is called. Acting upon that suspicion, we gave him salicylate of soda in full doses. His temperature came down with a run, and a few weeks later he had developed a definite cardiac bruit. This, then, was a case of acute rheumatic fever without any joint manifestations. Such cases are not common, but when they do occur it is very important that you should be able to recognize them, because if you allow rheumatism in a child to go on untreated for a few days, you run a great risk of endocarditis developing and permanent cardiac disease being the result.

There is in young infants a cause of acute fever which you should never overlook, and it is one which is especially apt to escape the observation of a physician, and that is **osteo-myelitis.** I have seen a child lie in the medical ward of a hospital for some days with acute fever, the cause of which was not explained. It died, and, post-mortem, one radius was found entirely surrounded by a layer of pus. That is not unlikely to happen to you in practice, and the moral is, when you have a case of acute fever with no other signs in a young child, you should always examine the bones with great care for signs of tenderness and swelling, always bearing in mind the possibility that the fever may be the result of acute suppurative periostitis or osteomyelitis. Another point of great importance is that in every case of acute fever you should examine the **throat** for tonsillitis, which is usually of the follicular sort. I have seen several cases where it was a difficult matter to determine the cause of the fever, but where the whole case was cleared up by looking down the child's throat. It should be done as a matter of routine in every case.

In older children **enteric fever** will certainly give you a good deal of trouble. I have known several errors in diagnosis made from not recognizing the possibility of enteric fever being the cause of the pyrexia. Such a thing happened to me the other day. I had a child in hospital who was admitted with a moderate degree of pyrexia. There were some bronchitic signs in the lungs, rather pronounced, but nothing more. The child looked tuberculous, and one concluded on the whole, seeing that there was no enlargement of spleen, and no spots or anything else, that it was probably tuberculosis, and we were prepared to treat it on that assumption. But as time went on one noticed that the temperature gradually mounted up, and then the spleen began to be palpable. The Widal reaction was tried, and definite clumping occurred at once. That, of course, made the diagnosis clear. Now, such a mistake as that is very easy to fall into. The recognition of enteric fever in young children is more difficult than it is in grown-up patients, and if you have fever for which you can find no other cause, even in the absence of enlargement of the spleen or other signs, bear in mind the possibility that you may have to do with a case of enteric fever, and by having the Widal reaction done, or by estimating the number of leucocytes, try to arrive at an exact diagnosis.

There are also other cases of fever of apparently intestinal origin besides the enteric cases. Amongst these are the so-called **food-fever,** which appears to be due to an excess of carbo-hydrates in the diet, along with constipation; cases of **para-typhoid** infection, and cases of **mucous colitis.** In all of these fever may be the chief or only sign of disease.

Acute tuberculosis may also cause high fever with no other physical signs until it terminates in tuberculous meningitis. I have already dealt with its diagnosis when speaking of tuberculosis in childhood.

There is a cause of fever in infancy which is of rare occurrence, but which it is extremely important that you should recognize when it does occur, and that is **acute pyelitis.** Last summer I was called in consultation to see a baby who presented some rather puzzling features. She was a girl of a few months old who had for some weeks been running a very high temperature with occasional remissions, and—this is one of the suspicious and important points—accompanied at times by definite rigors. On inquiry into the state of the urine it was found to contain pus. One then felt certain that it was a case of pyelitis. Further examination showed that the urine contained the *Bacillus coli communis*, and on giving the child alkalies in full doses, which is the right treatment in such cases, the temperature speedily came down.

INABILITY TO TALK.

Passing from pyrexia, I want to take up together two symptoms which mothers frequently complain of in their infants. One of these is *inability to talk* at the proper age, and the other is *inability to walk*. If a child is brought to you because of **inability to talk,** you should bear in mind that there are only a few possibilities which you need consider. In the first place, of course, the child may be a **deaf-mute.** In such a case you will find quite a clear history that the child is absolutely deaf, and you will sometimes find that there is a history of several similar cases having occurred in the same family. Such cases should not present any great difficulty in the matter of diagnosis. You find, again, that some children are unable to talk at the proper time because of **mental deficiency,** or else because of backwardness short of actual mental deficiency. These cases are more difficult to recognize. You should examine the child for the physical stigmata of mental deficiency, for cranial abnormalities, and the other signs which you know to be the outward expression of retarded development of the brain. But in the lesser degrees of mental deficiency where such physical signs are absent or are not pronounced, you may often be in doubt as to whether that is truly the cause of the child being backward in talking.

Pathological conditions in the throat, especially **adenoids** and **enlarged tonsils,** may be a cause of difficulty in learning to talk; I cannot exaggerate to you the importance of this fact. I should say that the great majority of children I have seen who were brought to me because they were unable to talk at the proper age were suffering from some abnormality of the throat. A typical case of this kind was that of a girl two years and three months old, who was brought by her mother because she was unable to talk, although 'she made violent efforts to do so.' The mother said the child heard well, she was obviously quite intelligent, and the only thing one could make out on physical examination was the presence of an enormous mass of adenoids in the naso-pharynx. These were removed by operation, and five months later the mother wrote that the child was talking well. That is a clear and typical instance of what I am referring to, and I could quote dozens of such cases almost which I have seen in the out-patient room here and elsewhere. It is difficult to say in such a case why it is that adenoids make it difficult for the child to learn to talk.

The mother is probably correct when she says that the child does hear, but the possibility is that he does not hear quite distinctly, not well enough at all events, for him to learn to imitate the speech of other people. Whether it is that, or whether the adenoids impose a mechanical obstacle to the movements of the soft palate I do not know, but I know if a child is brought to you with the complaint that he cannot talk, and you find adenoids present in large amount, you can promise with almost complete confidence that if the adenoids are removed the speech will become perfectly natural.

A rare cause of this lack of the ability to speak is **tongue-tie.** Mothers think it is a common affection, and most children who have difficulty in learning to talk are popularly believed to suffer from it. Now, tongue-tie does occur, but it is very rare. I can only recall at this moment three well-marked cases of it, and I doubt if it ever does produce true inability to talk—that is to say, real dumbness. It may cause difficulty in talking clearly and in pronouncing certain consonants for which the tongue is particularly required, but you must distinguish that from inability to talk at all. A still rarer cause of inability to talk is **congenital aphasia,** a condition of which I have never met with an example, but which is described as one of the occasional congenital defects of childhood.

INABILITY TO WALK.

Now, to turn to the kindred symptom—inability to walk. There you have to recognize, I think, two groups of cases: first, those in which the child has never walked at all from birth; and, second, those in which the child has at one time or another been able to walk, but has gone 'off his legs,' as the mother puts it.

First, with regard to those who have never walked. There you have three possibilities to deal with: either the child is the subject of rickets, or of some form of paralysis, or it is a case of mental deficiency. Now, in **rickets** the difficulty is merely one of walking. Whilst the child sits on his mother's lap he can move his legs about freely, whereas in cases of paralysis the child is not only unable to walk, but he cannot move the affected limbs even when sitting. In rickets you will find also the other general signs of the disorder—enlargement of the epiphyses, deformity of the chest, and so on; the knee-jerks also are unaffected, whereas in paralysis they are exaggerated or diminished.

Of **paralysis** as a cause of inability to walk there are two chief varieties: one is infantile paralysis, the other is spastic or cerebral paralysis. Infantile paralysis usually comes on definitely at some period subsequent to birth, whereas spastic paralysis is often congenital. In infantile paralysis you will find the knee-jerk on the affected side diminished and the muscles atrophied. In spastic paralysis you will find the knee-jerk exaggerated and the muscles of the limb well developed.

Mental deficiency you will recognize by the other general signs of mental impairment which I have mentioned before. Never forget, however, the possibility that children may be unable to walk from **surgical reasons** as well as medical. I have seen cases of *congenital double dislocation* of the hip produce great delay in learning to walk, and such cases are apt to be overlooked by the physician whose mind is fixed upon medical points.

When a child who has hitherto been able to walk 'goes off his legs,' you are dealing with a different order of cases. Many children who have had an **acute illness** will go off their legs for a long time after that illness, although you can find no special cause for their doing so. Probably it is partly because of muscular weakness, and partly because the child actually forgets how to walk; having only recently acquired the rather difficult art of walking, he gets out of practice when laid up in bed during an acute illness, and when he gets on to his feet once more he has to learn all over again. Such cases may go on without walking for a long time, but you may rest assured that they will walk in time. Another cause of a child going off his legs is that he may have developed infantile paralysis, but I have already told you how that is to be recognized.

A rarer cause which it is important to recognize is **rheu-**

matism, slight degrees of which affecting the hip or the muscles at the back of the knee-joint or ankle may cause the child to go off his legs on account of pain.

Remember, again, the possibility of surgical causes. One of the earliest signs of hip disease may be that the child goes off his feet.

HEADACHE.

The common causes of headache in childhood are not the same as in adult life. Circulatory disturbances, for instance, are rare in early life, whilst 'reflex' headache—particularly from 'eye-strain'—is frequent. If a child of school age suffers from headache, I should recommend you to examine his **refraction** and see if there is any hypermetropia or astigmatism, and if you find either to get the error corrected by proper glasses. If you do that you will often find that the headache disappears at once. Another group of causes of headache may be described as **toxic;** constipation and biliousness are examples, just as in grown-up people, and these are to be met by appropriate remedies. **Rheumatic headache** is not uncommon, the pain in this case being situated in the scalp. **Fatigue,** either physical or mental, may easily cause headache, especially in children who are growing fast. It yields rapidly to rest and tonics. **Cerebral tumour,** again, occurs with considerable frequency in children, and may be a cause of severe headache. Here you should inquire for other symptoms of tumour, such as vomiting, and look particularly for optic neuritis. **Adenoids** are a pretty common cause of headache in childhood. Here the headache is usually frontal. **Migraine** also is a disease of comparative frequency in children, in whom it occurs quite as often as in grown-up people, and frequently with great severity. Hysterical headache, on the other hand, is a condition of very rare occurrence in early life. Lastly, you should never omit to examine the urine in cases of headache, as an unsuspected **nephritis** which has perhaps followed one of the infectious fevers may be the cause.

CROUPY RESPIRATION.

A symptom of very great importance, and one which produces great anxiety in the minds of the friends, is that spoken of as laryngeal or croupy breathing. You are called to a child who exhibits croupy breathing, and you have to determine what the cause of it is. I need hardly say at the outset that the two things you will first think of, quite rightly, are so-called 'croup' on the one hand, and diphtheria on the other. Now, 'croup' usually means a **catarrhal laryngitis,** or it ought to mean that. Of this there are two sorts: first, that in which there is a slight degree of catarrh, but every now and then there is spasm superadded, producing croupy breathing; second, there are the more severe cases, in which the catarrh of the larynx is so great that it causes considerable swelling of the mucous membrane, and produces more or less continuous obstruction. The first sort of case you are not likely to confuse with **diphtheria,** because the history is that such a child is comparatively well during the day, but that during the night attacks of croupy breathing come on. The real difficulty arises in the second group of cases, in which the catarrh has led to so much swelling as to produce continuous obstruction. These are the cases in which it is difficult to tell whether the disease is diphtheria or not. In some cases, indeed, the diagnosis may be impossible. But there are certain points to which you should direct your attention. First, there is the mode of onset in the two cases. In catarrhal laryngitis the onset is usually more abrupt and severe than in diphtheria, which begins more insidiously. Catarrhal laryngitis is accompanied by a higher degree of fever. In the second place, the loss of voice and cough are less in catarrhal laryngitis than in diphtheria. In other words, diphtheria is the more *silent* disease of the two. If you find signs of a membrane on the fauces, of course you need have no difficulty at all; but the puzzling cases are those in which the diphtheritic process is confined to the larynx only, and although the above indications may be of some help, every now and then you are bound to be left in doubt. In such a case the wisest plan is to act as if it were the more serious disease—that is to say, isolate the patient and give antitoxin.

But there are other causes of croupy or laryngeal breathing which it is well to bear in mind. It may occur, for instance, as a result of **retro-pharyngeal abscess,** and I have known serious mistakes arise in this way. If in doubt, put your finger into the child's throat and explore the pharynx, and see if you can find any indication of bulging. Croupy breathing may also occur in the course of a **chronic laryngitis,** which may be simply catarrhal or the result of congenital syphilis. It may occur also from pressure on the trachea, by **enlarged bronchial glands,** or, in rare instances, by an **enlarged thymus.** It may also be simulated by certain conditions, the most notable of which are **congenital laryngeal stridor** and **laryngismus stridulus,** although there the nature of the attacks should leave you no doubt. Congenital laryngeal stridor dates from birth, whilst laryngismus stridulus is always preceded by a period of apnœa, followed by a long crowing inspiration, which does not resemble the continuous croupy breathing of laryngeal obstruction. It is always well also to remember the possibility that you may be dealing with a **papilloma of the larynx**. Two or three years ago a baby was brought here in my absence and seen by my assistant. The child had continuous croupy breathing, and the question arose, What was the cause of it ? The symptom had been noticed since birth, and it was rightly thought that it might be a case of congenital larnygeal stridor. The child died suddenly within a week, and the post-mortem examination revealed the presence of laryngeal papilloma. I have since seen two cases where there was continuous slight croupiness of breathing, and in which removal of a papilloma caused the disappearance of the croup. Lastly, bear in mind always the possibility that croupy breathing may be the result of inhaling a **foreign body.** Careful attention to the history will generally keep you right here, because the onset in such a case is always, of course, extremely sudden.

SCREAMING.

Constant screaming is a distressing and not infrequent symptom in young children for which you will sometimes be consulted. If you have excluded **hunger** and **temper** as causes, you may regard it as a sign of pain, and it will be well for you to keep clearly before your minds the chief causes of such pain, so that you may be able to make a correct diagnosis, as you sometimes must do, by a method of exclusion alone.

Certainly the commonest cause of pain which results in screaming in an infant is **colic.** You will recognize it by the fact that the screaming is accompanied by drawing up of the thighs upon the abdomen and kicking of the feet. The motions, too, are usually abnormal in one way or another. Sometimes the mother or nurse will be able to tell you that the screaming ceases with the passage of flatus from the bowel, and if you get such a history the diagnosis cannot be in doubt.

Renal colic, which is far from infrequent in childhood, may simulate intestinal colic very closely. It is rarely, however, so persistent, and you will be able to recognize grains of uric acid on the napkins. If the screaming is specially associated with the act of micturition you should suspect **hyperacidity of the urine,** or, in boys, **phimosis.**

Earache is another pretty frequent cause of screaming, but usually it will have been noticed that the child put his hand up to his head when the pain and screaming came on. Infants who are the subjects of **scurvy** always scream a great deal. Here the screaming tends to come on with movement, and one leg or both is kept as still as possible—quite different from the restlessness and kicking of colic. The screaming of scurvy may be simulated by that due to the **hyperæsthesia** which is often present in the early stages **of infantile paralysis** (see p. 258), but the absence of any thickening of the limb in the latter condition should keep you right. The screaming of early **meningitis** is not likely to lead to error, for it is of a peculiar and penetrating character, and is accompanied by more or less of head retraction and other signs of irritation in the meninges.

Lastly, if you can find no other cause for the screaming, it is always well to remember the possibility of **mental deficiency,** for some mentally defective children exhibit persistent screaming almost from birth without there being anything one can discover to account for it.

INSOMNIA.

Sleeplessness is a not uncommon trouble in early life, and one which is a great source of anxiety to parents. In some children it seems to be almost a congenital peculiarity. You will be told that 'the child has never slept well.' In such a case you will usually find that there is an inherent instability of the nervous system which shows itself in general excitability and nervousness.

Apart from such congenital predisposition the commonest exciting causes of insomnia in young infants are indigestion and teething. In every such case, therefore, it is your duty to inquire carefully into the feeding, into the possibility of colic, and into the state of the bowels. By rectifying anything which is wrong in these directions you will often effect an immediate cure. In other cases, again, insufficiency of food is the cause, and sleep is restored when more food is given. Much also seems to depend upon the personality of the nurse. Some nurses seem to have the knack of getting infants to sleep well, whilst others have not ; it would almost appear, indeed, as if in certain cases the nurse got on the infant's nerves, so remarkable may be the effect of a change of attendant. In persistent and intractable cases of insomnia in infancy one is always rather suspicious of mental defect, of which sleeplessness seems to be an occasional symptom.

In older children the two great causes of bad sleeping are—(1) *indigestion*, and (2) *overstrain at school*. Obstructed respiration from adenoids or large tonsils is also a potent cause. The treatment to be adopted in each of these cases is self-evident. Hypnotics should only be resorted to temporarily and in order to aid in the re-establishment of the sleep-habit. Bromural is one of the best. It may be given in 5-grain doses with a little hot milk at bedtime to a child of five. Avoidance of all excitement, an open-air holiday, and attention to the digestion and bowels are, however, the sovereign remedies.

SWELLING OF THE ABDOMEN.

Enlargement of the abdomen is a symptom for which children will sometimes be brought to you. The first thing to bear in mind here is that the abdomen of young infants is normally rather prominent, and an anxious and inexperienced mother is apt to mistake the natural prominence for a sign of disease (see p. 6). Assuming that you have satisfied yourself that the prominence is really pathological, you should look next for signs of **rickets,** bearing in mind how greatly the abdomen may be enlarged in that disease. The reasons for this I have already pointed out (p. 136).

Tuberculous peritonitis is another frequent cause of abdominal enlargement in children of two years and upwards, and should be examined for with great care. In the exudative form the discovery of ascites will determine the diagnosis, whilst in the adhesive variety one must examine carefully for lumps or thickened omentum. Mere **chronic intestinal dyspepsia** with flatulence may give rise to considerable and lasting abdominal distension, but I think it is a good rule to be suspicious of tuberculosis in any such case in which appropriate treatment does not effect a speedy improvement.

Enlargement of the viscera, particularly of the spleen or kidney, may cause a general abdominal prominence. I need not remind you of the main points to be attended to in distinguishing a large spleen from a large kidney, but would only say here that their differentiation is by no means always so easy as the text-books might lead you to suppose, and that mistakes in the matter are apt to be made even by the most experienced and careful. The chief causes of enlargement of the kidney in early life are sarcoma and hydronephrosis. Great enlargement of the spleen is usually associated in infancy with anæmia (see Splenic Anæmia, p. 309), and in later childhood has probably often a syphilitic origin. Myeloid leukæmia is an *extremely* rare cause of large spleen in the child, and malaria, in this country, is also a very uncommon occurrence.

JAUNDICE.

The last of the symptoms to which I wish to direct your attention to-day is jaundice. This may be a sign of several different diseases of the liver, the differential diagnosis of which is often a matter of no small difficulty. It will be convenient for us to consider the case of infants and of older children separately.

The majority of infants suffer, as you know, from a slight degree of jaundice coming on a few days after birth, to which the term **icterus neonatorum** is applied. Usually this is but a trivial affair, lasting only for a week or so, but occasionally it may be prolonged for several weeks, and in such a case your diagnosis may be in doubt. What the cause of this unusually severe development of icterus neonatorum may be I cannot tell you, but it is interesting to note that in at least two cases I have known it to be followed by severe and persistent anæmia. Another, but very rare, cause of jaundice coming at or soon after birth is **congenital obliteration of the bile-ducts.** Usually you will have no difficulty in recognizing this condition, for it is characterized by a very severe degree of icterus with complete absence of bile from the stools. The liver, too, is notably enlarged, hard, and irregular, and the spleen is palpable. It is a serious condition, and usually proves fatal before the end of the second month.

Syphilitic hepatitis—a small-celled infiltration of the liver—is a third and not uncommon cause of jaundice in infancy. It is not always easy to tell it from a severe case of icterus neonatorum, but other signs of congenital syphilis are usually present, and the liver is definitely enlarged. Whether it is ever recovered from I cannot say for certain, but I think I have seen cases clear up under mercury. In older children by far the commonest form of jaundice is the **catarrhal** variety. Do not be surprised in such cases to find the liver enlarged. It usually is so, reaching, perhaps, two or three fingers' breadths below the edge of the ribs, but rarely further.

Sometimes it is slightly tender as well. **Hepatic cirrhosis** is the condition from which you have to diagnose catarrhal jaundice, and it is not always easy to do so. The icterus in such a case, however, is usually but slight, and there is a prolonged history. Enlargement of the spleen, too, is the rule in cirrhosis, whereas it is absent in the catarrhal form.

Of the less frequent forms of jaundice, such, for example, as occur in the course of **infective processes,** of **acute yellow atrophy,** and other rare diseases, I have not time to speak in this lecture.

LECTURE XXVIII

SOME COMMON SYMPTOMS OF DISEASE IN CHILDREN AND THEIR DIAGNOSTIC SIGNIFICANCE—*Continued*

GENTLEMEN,—In my last lecture I took up some aspects of disease in children from what one may describe as the symptomatic standpoint ; that is to say, I selected certain common symptoms, and then tried to make out what their diagnostic significance was. I believe that to be an exceedingly important way of treating a subject. It is essential for you to have your medical knowledge 'indexed both ways' ; in other words, you should be able at examinations, given a certain disease, to tell what are its symptoms, and you should be able at the bedside, given certain symptoms, to say what is the disease. The training which fits you to reply to the one set of questions does not always enable you to reply to the other. I make no apology, therefore, for taking up some more symptoms in the same manner, and trying to arrive at an idea of their diagnostic value, and shall deal to-day with some symptoms relating to the alimentary system.

ABDOMINAL PAIN.

Abdominal pain is a very common symptom in children, and its cause is often difficult to determine, because if there be, as there often are, no objective signs of disease, you have very little to go upon in forming your diagnosis. A child is unable to tell you exactly where the pain is, when it comes on, what is its character, and so forth ; and hence you find lacking in children those guides to diagnosis which are often present in the case of abdominal pain in grown-up persons. There is all the more reason, therefore, why you should study carefully what the possible causes of abdominal pain are, so that you may proceed to your diagnosis by the method of exclusion.

The first thing I would remark about abdominal pain in childhood is that it may be due to causes which are outside the abdomen altogether. I would therefore group the first set of causes as *extra-abdominal*—that is to say, cases in which pain is referred to in the abdomen, although its site of production does not really lie in the abdominal cavity at all. One fairly common example of such an extra-abdominal cause of pain is **spinal caries.** The pain of spinal caries, travelling along the intercostal nerves, is very often referred by the child to the epigastrium, and the first piece of advice I would give you is that when pain is complained of in the epigastrium you should never omit carefully to examine the vertebral column for signs of disease. Another extra-abdominal cause of pain is **pleurisy.** Dry pleurisy is not common in children, but when it does occur the child often refers the pain to the pit of the stomach. It may therefore be necessary, in order to exclude this cause, to examine the bases of the lungs carefully for friction sounds, because it is only when the pleurisy is of the dry variety that sufficient pain is produced to give rise to symptoms. Another possible cause of pain referred to the abdomen, but really due to causes outside it, is **hip disease.** In a few cases the pain of hip disease is referred by the child to the iliac fossa, and you are apt to be misled, and to think you are dealing with appendix mischief or something of that sort, when all the time the seat of the trouble is in the hip. You may have to satisfy yourself as to the absence of all these causes before you conclude that the pain is truly intra-abdominal in character. Before leaving this part of the subject I should mention that abdominal pain seems sometimes to be of **rheumatic** origin, although it is uncertain whether its seat in such a case be really intra-abdominal or merely in the abdominal wall. The pain comes on paroxysmally, starting usually below the costal margin on either side, and lasting for a few minutes. There may be several such attacks in the course of the day, and, after lasting for a few days, they may culminate in acute rheumatism.

Passing now to the causes of abdominal pain which are due to disease *inside the abdominal cavity*, I would point out that pain of intestinal origin is certainly commoner in children than pain of gastric origin. In other words, any pain in the abdomen in a child is more likely to be due to something wrong with the intestine than to some disorder of the stomach. The reason is that children do not often suffer much from organic diseases of the stomach. Functional dyspepsia, I need hardly say, they do suffer from to a great extent ; but such organic diseases as ulcer or carcinoma of the stomach children do not exhibit, except in rare cases. Now, you know that functional dyspepsia does not give rise to severe pain ; it causes discomfort, distension, sensations of sinking, and so on, but not real pain. With regard to intestinal pain, I should say that the most common cause of it is **colic,** in one or other form. Such intestinal pain is often very deceptive, for the reason that it may come on immediately after the taking of food. You will constantly see children—they are usually about five to ten years of age—who are brought with the complaint that there is pain in the abdomen which comes on immediately after eating, and you will naturally think that the pain must be due to some condition in the stomach itself. But you must not allow yourself to be deceived in that way, because it would seem that the entrance of food into the stomach often excites peristaltic contractions in the intestine, particularly, perhaps, in the colon, which contractions give rise in certain patients to a pain which is colicky in character. The most conspicuous example of such excitation of peristalsis in the large bowel is found in the case of lienteric diarrhœa. In that condition, as soon as the child takes food there is an immediate tendency for an action of the bowels to occur. The food has excited a peristaltic contraction in the colon, and that leads to defæcation. Similarly, in certain cases, the entrance of food into the stomach gives rise to contractions in the bowel such as produce colicky pain. Sometimes a very small amount of food will cause it. I have even known cases in which the taking of a little milk gave rise to severe distress. The pain may be so severe, for instance, that the child has to rise from the table and lean against some hard object, such as the back of a chair, in order to get relief. Further, you will find that in many cases the kind of food that the child eats does influence the pain, that it is worse after eating hard and comparatively indigestible foods. I suppose that is because such bodies stimulate the interior of the stomach much more powerfully than the blander forms of nourishment do, and so give rise to more powerful reflex impulses. At any rate, all these things taken together—the fact that the pain comes on immediately after eating, and that it is influenced by the nature of the food—are apt to lead you into error, and make you think it cannot be pain of intestinal origin, but that it must be due to some disorder of the stomach. But I advise you not to be so deceived, and to remember the possibility that you are dealing with a purely intestinal pain. Another point about these intestinal colicky pains in children is that they frequently come on when the child walks quickly. Perhaps the commonest circumstance in which they come on is hurrying off to school after breakfast ; such a pain is usually referred to the right iliac fossa, and may be so severe as to necessitate sitting down. I do not pretend to be able to explain why it is that exertion brings on pain of that sort, but I think most people suffer occasionally in that way, and must have experienced the fact that violent exertion, or even hurrying after a meal, is apt to produce what is popularly called 'a stitch in the side,' usually on the right side, in the region of the cæcum.

Chronic appendicitis may be a cause of abdominal pain. The position of the pain will draw your attention to its possible cause. In trying to confirm or exclude this cause, you will have to palpate the appendix region with special care, and note whether there is a point of special tenderness between the umbilicus and the anterior superior spine on the right side—that is to say, over the site of the appendix. If you find there is a tender spot there, and particularly if the child has recurrent febrile attacks, your suspicion that it is appendicitis will have been confirmed.

Another cause of occasional pain in the intestines undoubtedly is the presence of **worms**—particularly, I think, of round-worms. Now, it would be a mistake to suppose that to be a common cause; still, one does every now and then meet with cases in which a child has suffered from intestinal pain, often referred to the region of the umbilicus, in which relief has only been experienced after the passage of a round-worm, either by the bowel or by vomiting. And I think one is bound to conclude that such a cause may account for the pain, and that the worms have sometimes acted in the way in which a lump of indigestible food would act.

Passing from these intestinal causes, one has to mention another possible source of abdominal pain which you are apt to overlook—namely, pain which is produced **in the urinary tract.** By that I mean the pain which arises on the passage of uric acid or small stones down the ureters. The pain due to this cause may closely simulate intestinal colic. It comes on irregularly; it is referred usually, if the child be able to refer it to any spot, to the lateral region of the abdomen, and it often passes down into the groin. An X-ray examination of the kidney and ureter and bladder will often be of great help in such a case.

There remains, lastly, for consideration the pain that is of **gastric origin.** I will put a query against that on the board, because I am not certain whether pain of gastric origin occurs at all in childhood. Still, one does meet with cases where the results of treatment seem to show that the pain has been of this nature—cases in which the child has complained of pain, as a grown-up person does, a short time after food, perhaps one to two hours afterwards, and where the pain has been relieved by giving drugs which are directed to exert either a soothing effect upon the stomach or to neutralize hyperacidity. I had such a case not long ago in the person of a little girl who complained of pain in the stomach shortly after meals. Examination revealed nothing definite, but I treated her with carbonate of magnesia, and the pain at once disappeared. I think there is reason to suppose that such pain is due to the overproduction of acid, just as may happen in an adult. But I believe such cases are uncommon in children, and in the majority of them you will be wise to suspect that pain arising soon after meals is not of gastric but of intestinal origin, and inasmuch as colicky pains are more common, it is of their **treatment** that I wish more particularly to speak to-day.

The first thing to be done in dealing with intestinal colic is to see that the diet is adjusted in such a way as not to produce much fermentation in the intestine, and you should therefore see that the food does not contain those ingredients which tend to produce large quantities of gas—I mean such articles of food as are rich in cellulose, which is the source of marsh gas in the intestine. You will therefore eliminate green vegetables, restrict or abolish the consumption of fruits, and limit all the starchy things in general, whilst you may have to increase the amount of animal food in the diet so as to compensate for the restrictions in other directions, and insure that the child is sufficiently nourished. So much for the dietetic treatment.

Now, there is no doubt that some of those cases are aggravated, if not actually caused, by chilling of the abdomen. It is always wise, therefore, to order that the child should wear a warm flannel abdominal binder. Lastly, you will have to consider the question of drugs. Many—perhaps, indeed, the majority—of these patients suffer from constipation. Even when there is no apparent constipation it is advisable to begin the treatment by administering laxatives, and it is well to combine with these such drugs as belladonna or hyoscyamus, which have the power of diminishing intestinal spasm. I have found a combination of cascara and belladonna in the following form very useful: Fluid extract of cascara 5 to 10 minims (the dose being graduated according to the condition of the bowel), tincture of belladonna 5 minims, aromatic spirits of ammonia 15 minims, syrup of ginger ½ drachm, and peppermint water to a couple of drachms. The child takes this dose three or four times a day, after meals. You will find that the effect of a medicine of that sort is to cause a gentle laxative action, and, in addition, belladonna tends to prevent any griping, and with some such treatment as that you will, in the majority of your cases, meet with success. Sometimes, however, you will find that the pains persist in spite of all you can do, and if you are face to face with a case of that sort you may be compelled to fall back upon opium. Two grains of Dover's powder night and morning, or 2 grains three times a day if necessary, will be found sufficient in most cases to allay the pain. The disadvantage of opium is its constipating tendency, and the fact that it has often a depressing effect upon the digestion. But it is curious that where opium is needed, and where it is doing good, the constipating tendency is often in abeyance. The opium seems to act in such cases simply by relieving the irregular peristalsis and spasm of the bowel, and not by causing a cessation of the peristalsis altogether.

DYSPHAGIA.

I want now to pass on to speak briefly of another symptom, one which is far less common, though of considerable interest, and which will sometimes puzzle you and occasion you a good deal of trouble in practice ; that is, dysphagia in children. Difficulty in swallowing is a subject which you do not read much about in books which deal with diseases of children, but you will find that it is not a very uncommon occurrence in little babies and in children up to, though not often beyond, the age of three. By dysphagia I mean a difficulty in carrying out the act of swallowing. The mother will tell you that the child takes a mouthful of milk, and when it gets to the back of the throat it seems to lodge there ; the child makes an abortive attempt at deglutition, but the milk comes back again out of the mouth, very little going down the throat. Your advice is sought as to the cause of this, and how it is to be put right. The first thing to do in a case of this sort is to make sure that there is no mechanical obstruction in the throat. The most common **mechanical obstacles** are *congenital adenoids*—which I have repeatedly known to cause difficulty in swallowing (p. 368) —and *cleft-palate.* I saw not long ago a baby in whom this symptom was very well marked. For a time it puzzled me. I examined the throat by rather an indifferent light, and I thought I saw nothing wrong, and I could not understand what the difficulty in swallowing was due to. But next time the child was brought to me I had a better light, and I saw that there was a bifid uvula. I have no doubt that was a sufficient cause for the difficulty in swallowing in this case. You will frequently find difficulty in swallowing in children who suffer from **mental deficiency.** The act of swallowing, like most other co-ordinated muscular acts, is acquired with difficulty by these children, and if you can see no local cause for the dysphagia you should bear in mind the possibility that you are dealing with a child who is mentally defective. In older children you may find difficulty in swallowing result from **paralysis of the palate,** which may be one of the sequelæ of diphtheria. It may also be seen in children in whom the throat is very irritable and congested, and where there is some enlargement of the tonsils. You may find in such a case that as soon as a crumb or anything solid gets into the child's throat it excites hawking and retching movements, and the child cannot swallow it. Some of these cases are exceedingly troublesome, and I remember one in which the use of purely liquid food had to be persisted in so long that the child became rickety. In some cases difficulty in swallowing is due, I believe, to what one must describe as **perverseness** on the part of the child, or pure 'cussedness.' Many children who are accustomed to the bottle, especially if they have been allowed to have it too long, very much resent the transition to solid food, and they show their resentment by hawking and coughing, and spitting out any solid food which is given to them. I had a case in private the other day where the mother was very alarmed at this difficulty. The only plan in such circumstances is firmness, to insist again and again, no matter how often the food is rejected, on the child taking it. Sometimes you can overcome the difficulty by gradually increasing the consistence of the food ; you can begin by thickening milk with farinaceous material, and get it gradually thicker, until it is practically solid. At any rate, I am sure that perseverance in such cases can always overcome the trouble.

VOMITING.

I have already considered chronic vomiting **as it occurs in infants** in a previous lecture, and would only remind you here that you have to distinguish between (1) 'physiological' vomiting, the automatic rejection of excess of milk, or what the nurses sometimes call 'puking,' the distinctive feature of which is that it is not attended by loss of weight; (2) the vomiting of pyloric spasm; (3) the vomiting of indigestion. The differential diagnosis of these is fully considered in Lecture IV.

After the period of infancy **acute vomiting** is much more often met with than chronic. It may be due (1) to *acute indigestion*. The history of the taking of an excessive or indigestible meal, or of some special article of food, will usually keep you right here. The vomiting of acute indigestion is not accompanied by fever, but in (2) *acute gastritis* this may be present. Here, again, there will usually be a history of some noxious substance having been swallowed, or of the exposure to cold; but sometimes you will have difficulty in distinguishing it from (3) *symptomatic vomiting*, which sets in at the outset of some acute disease. Any of the acute specific fevers may be so ushered in, especially scarlet fever, but vomiting is also a very frequent symptom at the outset of pneumonia. *Meningitis*, again, is often preceded by vomiting for a short time before any other signs develop. A combination of vomiting with a slow pulse and constipation is very suggestive of it. (4) *Acute intestinal obstruction and appendicitis* should always be thought of in a case of sudden vomiting, especially if attended by abdominal pain. Remember, too, the comparative frequency of *cerebral tumour* in childhood, and in all cases of chronic vomiting examine the optic discs. *Uræmic vomiting* is not common in children, but there is no harm in examining the urine in an obscure case. Gastric ulcer and malignant disease, which so often cause vomiting in the adult, are very rarely met with in childhood.

Recurrent attacks of vomiting are sometimes rather puzzling. They may be due to so-called **'bilious attacks,'** which are closely allied to migraine, and may, indeed, be accompanied by severe headache. Such cases are almost indistinguishable from the form known as ‘ **cyclical vomiting.**’ This is a form of vomiting met with in nervous children, and in which the attacks recur at more or less regular intervals. During the attack the child may be quite prostrate, and utterly unable to retain anything—even water—on the stomach. The pathology of such cases is obscure, and it is uncertain whether the nervous system or the liver plays the chief part in their causation—perhaps both are concerned. In severe cases acetone may be present in the urine, and the odour of it recognized in the breath, but whether it is the cause or the consequence of the vomiting is disputed.

As I have not spoken of such cases before, I may add a word on their treatment. During the attack there is no use in attempting to give food by the mouth. The child should be kept warm, and a pint of saline solution injected into the bowel night and morning. In one case the vomiting persisted so long that we were driven to give food by the stomach-tube, by which method we succeeded in getting a fair quantity retained, and, I believe, saved the child's life. Morphia should be given subcutaneously in severe cases to prevent exhaustion, and stimulants, such as caffeine and strychnine, used if indications arise. An attempt should be made to open the bowels by calomel, but the constipation during the attack is apt to be extreme, and sometimes even large enemata are powerless to effect an action. If there be acetone bodies in the urine, as shown by a port-wine reaction to ferric chloride, bicarbonate of soda should be given freely, either by the mouth dissolved in iced soda-water, or, if it cannot be retained that way, by the rectum.

In order to prevent recurrence of attacks the child should live a quiet, regular, open-air life, all excitement and any overstrain in lessons being avoided ; sugars, starches, and milk should be cut down in the diet, and regular aperients administered, mercurials being specially useful. A judicious use of the bromides is also of help in very excitable children.

Hæmatemesis is not commonly met with in children, for the usual causes of it in the adult—cirrhosis, gastric ulcer, and malignant disease—are all very rare in the child. If, therefore, a child vomits blood there is always a strong presumption that the blood has been swallowed. In the case of an infant at the breast the blood may be derived from a crack in the nipple, and if you are in doubt as to whether this be really the source try the effect of a breast-pump. I have also known the blood come from the sucking of an inflamed gum during teething. True hæmatemesis may occur as a symptom of the hæmorrhagic disease of the newly-born (see p. 20), and I have also known it take place during the violent vomiting associated with pyloric spasm. In such cases, however, it is usually of the grumous or coffee-grounds variety—not profuse. In older children hæmatemesis is usually a symptom of a blood-state such as purpura or leukæmia, but occasionally it results from cirrhosis of the liver, and is then a serious symptom. Very rarely is it due to ulceration of the stomach.

ANOREXIA.

The last symptom I propose to consider with you to-day is loss of appetite, or anorexia. A good appetite is so natural to a healthy child that any impairment of it at once attracts the mother's attention, and children will often be brought to you for this and nothing else. In little babies loss of appetite may occur as a temporary matter from **anything which upsets the child's health.** Vaccination and teething are examples in point. In bottle-fed babies the cause may be **monotony in the meals,** and the introduction of a little variety, such as substituting a broth feed for one of milk, or the addition of a little Mellin's Food to some of the bottles, may set matters right. During the process of **weaning,** again, there may be difficulty in getting the child to take its food, whether the weaning be from the breast or the bottle. Here a little firmness is what is chiefly needed.

During the second year of life loss of appetite may occur in association with **chronic catarrh of the bowel,** a condition which I have considered at length in an earlier lecture (p. 78), and if the child be also rickety, as it often is, may constitute a serious obstacle to treatment. The treatment is that of the bowel condition. At this age, too, profound anorexia may precede the development of **tuberculosis,** and is therefore a symptom fraught with some anxiety.

In older children loss of appetite is a common symptom of **chronic dyspepsia,** a condition which we studied in detail in Lecture XIII. It may, however, be of nervous origin. Thus I have known it occur as a manifestation of **hysteria,** and only yield to isolation and the threat of the stomach-tube. I remember, too, a little boy of about four who was almost certainly somewhat of a ‘ moral imbecile ’ who flatly refused to touch any liquid out of a cup, and even preferred to be fed nasally, yet who would eat solids quite well. Such a case is difficult to classify. Occasionally, although very rarely, one meets with refusal of food from **melancholia,** just as one does more commonly in grown-up patients. I recall such a case in the person of a little girl of six who suffered from severe valvular disease, and who declined to eat ‘ because she did not want to get better.’ Such an unchildlike attitude, however, is fortunately very rare at this time of life.

As regards the treatment of loss of appetite *per se*, and in the absence of any organic disease, you should try the usual alkalies and bitters before meals, along with aperients and mercurials. If these fail there is nothing to equal change of air, especially to the seaside, in bringing back the natural desire for food. This is a remedy which is equally applicable at all ages.

LECTURE XXIX

SOME MEDICAL ASPECTS OF ADENOID VEGETATIONS IN INFANCY AND CHILDHOOD

GENTLEMEN,—I wish to direct your attention to-day to some medical aspects of adenoid vegetations as exhibited in the case of infants and young children. I shall assume that you are familiar with the local symptoms which are due to the presence of adenoid growths, and also with the chief surgical complications to which they may give rise—such, for example, as otitis media and other secondary suppurative processes—and shall confine my remarks more to those diseases which accompany adenoids, and which, more strictly speaking, come within the cognizance of the physician. I do not propose to deal at all with the pathology of adenoids, because that is at present very ill understood, and I can add nothing to what you already know on that aspect of the subject.

ADENOIDS IN INFANTS.

It will be convenient, I think, to divide the subject, first of all, into adenoids as occurring in infancy on the one hand, and in older children on the other. Perhaps it may be a surprise to some of you to hear that adenoids occur in infancy at all. Everyone knows that adenoids occur in older children. But I go further, and say there is reason to believe that adenoids may be a **congenital** abnormality—that is to say, that a child may be born into the world with adenoid growths in its naso-pharynx. In order to realize the **symptoms** that adenoids may produce in young children, our best plan will be to look at the clinical history of some cases which I have had under observation. I shall begin, in the first place, with the simplest symptoms to which adenoids may give rise in infancy. For instance, a child of five months old was brought to me with a history that it had been **snuffling at the nose** and had difficulty in breathing since the time it was born. The family history was perfectly good, and there was no suspicion of congenital syphilis. On examination the respiration was laboured, and the lower interspaces were drawn in during inspiration. The child had also a markedly adenoid facies. On examining the naso-pharynx one found quite easily a mass of fibrous adenoids, and after their removal the child was brought back with almost complete relief from all its symptoms. That would be one of the simplest cases of adenoids in infancy, and you will observe that it occurred in a child as young as five months. I have seen another case in a baby of nine months which was brought on account of discharge from the nose, accompanied by occasional **epistaxis.** This child had also a slight bronchitis, which is a very common accompaniment of adenoids and marked vegetations in the naso-pharynx. In that case, too, the epistaxis and the other symptoms disappeared after the naso-pharynx had been cleared out.

Another group of cases show, as their chief symptom, **cough.** A child only two months old was brought to me on account of a choking cough, which came on in paroxysms. (That is worth noting: it is the typical sort of cough which is associated with adenoids in the naso-pharynx. On account of this cough some of the patients with adenoids are supposed to be suffering from whooping-cough.) Further, the cough in this child was invariably accompanied by the involuntary passage of fæces, owing, I suppose, to the extreme spasm which it caused. Examination of the child showed the lungs to be perfectly healthy, but the naso-pharynx was full of adenoid growths.

Another baby had **snoring respiration,** and was only able to sleep in snatches, out of which he started up in a condition of partial asphyxia. During the day he also suffered from paroxysms of choking cough, and sweated profusely about the head, which is another symptom not infrequently associated with adenoid growths in infancy. In this case also great and immediate relief followed operation.

Another class of case exhibits **stridulous breathing** as the chief characteristic. A baby eighteen months old was brought to me for a 'catch in the breath,' which came on at intervals during the day and was also present during sleep. The veins of the head and scalp were distended, particularly at night, and there were moist sounds in the lungs. The child had enlarged tonsils and adenoids. I subsequently heard that he died suddenly a day or two after I saw him, and before operation was performed. I have known that occur in another instance, but in the majority of cases one finds that the stridulous respiration is much slighter in degree, and the children after having been submitted to operation make a good recovery.

Another class of case is that in which the child has **difficulty in swallowing.** A child sixteen months old was brought on account of difficulty in swallowing, this difficulty having been observed for a long time. The trouble was found to be greatest in the case of liquids, and on that account the child experienced difficulty in taking the bottle and had to be fed by means of a spoon, and then the fluid tended to run down the side of the mouth unless the head was held very much back. Examination showed that the tonsils were enlarged, and there was a mass of adenoids in the naso-pharynx. The throat was operated upon, and when the child was seen two months later the mother reported that there was very great improvement. Another child, three years and six months old, h[illegible] culty in swallowing, which dated from a[illegible] one year previously. The patient suffered [illegible] ness. The naso-pharynx was full of ade[illegible] was some enlargement of the tonsils. That child h[illegible] operated upon, but I do not yet know to what exte[illegible] symptoms have improved.

You will see from a recital of these cases that the **symptoms of adenoid growths in infancy** may be very various. They may be divided into three groups. First, there are those in which the chief complaint is snuffling and nasal discharge, occasionally accompanied by epistaxis. The second group is that in which the chief complaint is dyspnœa, often paroxysmal in character, and the breathing is definitely stridulous, frequently accompanied by attacks of choking cough. The third group comprises those in which the difficulty complained of is associated with swallowing, and especially with the swallowing of liquids.

The presence of adenoids in each of these groups may simulate other medical diseases, and it is on that account that they are particularly interesting to the physician. For instance, where there is nasal discharge, snuffling, and epistaxis, one has to distinguish between adenoids and congenital syphilis. You know that one of the symptoms of congenital syphilis is snuffling and the discharge of ichorous fluid from the nose, and there may occasionally be a little bleeding. Under such circumstances it is often difficult to tell whether the discharge and snuffling are due to congenital syphilis or to the presence of adenoids in the naso-pharynx. The only way to settle the matter is to make careful inquiry into the family history and to look for other signs of congenital syphilis besides those in the nose. In addition, I think the discharge from the nose due to the presence of adenoids begins sooner after birth than it does from congenital syphilis. You may know that there is a distinct interval between birth and the appearance of the discharge in congenital syphilis, and sometimes the interval amounts to several weeks. But the snuffling and discharge from the presence of adenoids may begin immediately after birth. You might think that a local examination of the naso-pharynx ought to settle the matter, but, unfortunately, it is difficult to examine the naso-pharynx of children satisfactorily. If any of you have tried it you must have noticed that there is an extremely small space between the soft palate and the posterior wall of the pharynx in a young infant, so that you can hardly get your finger up behind the nose. So one can only infer the presence of adenoids in them from the symptoms; one cannot, as is the case with older children and adults, make certain of it by digital examination. Anything like posterior rhinoscopy, I need hardly say, is quite impossible in young infants.

Another medical disease which may be simulated by these growths is the condition known as **congenital laryngeal stridor,** which I have described to you before (p. 214). How the presence of congenital adenoids leads to the stridulous respiration I am not prepared to say, but there can be no doubt whatever that congenital adenoids do lead to that, for not only is it illustrated by the cases I have mentioned, but many other observers have recorded a similar condition. There was a case reported some time ago in the *Lancet** by Dr. Eustace Smith, in which the symptoms disappeared immediately after the removal of the adenoids.†

Now we come to the third group, those in which the chief difficulty is that of swallowing. These are an interesting group, as they may lead to serious malnutrition. I have seen cases of marked wasting in young infants brought about by this **difficulty in swallowing** milk. The child is unable to suck the bottle, and has to be fed by the spoon, and that is often found inadequate. The mechanism of these cases is easy to understand. When the naso-pharynx is blocked by a mass of adenoids it is impossible for the child to breathe through the nose while sucking. He has only the mouth to both breathe and suck by, and consequently, when he is using the mouth for sucking he cannot be using it for breathing, and gets short of breath, and thereupon instantly ceases to suck. Accordingly, he gives up the bottle in despair, and all attempts to feed such a child by the bottle end in failure. Even the feeding by means of a spoon in such cases may be difficult, because I think the presence of adenoids interferes mechanically with swallowing—that is to say, not merely through the respiration, but by opposing mechanical obstacles to the act of deglutition. One way in which you can imagine that happening is that the mass of adenoids may prevent the elevation of the soft palate into the naso-pharynx, which is a normal occurrence during healthy deglutition. This difficulty in swallowing may sometimes be extremely marked. I remember a case which was brought to this hospital, and was under the care of one of my colleagues, in which the difficulty was so marked that the child could only swallow when it held its head very far back, like a bird. You have, perhaps, noticed that when a bird drinks it takes a mouthful and then holds its head back in order to swallow it. In the case of this child other children had given it things to drink so as to have the fun of watching it throw its head back in order to swallow them. That child had a considerable growth of adenoids, and the condition improved very much after they were removed.

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* 1898, i., p. 783.

† See also Lack (*Journal of Laryngology*, 1898, xiii., p. 303); Lubet Barbon (*Revue des Maladies de l'Enfance*, 1891, ix., p. 499); Huber (*Archives of Pediatrics*, 1894, xi., p. 38); and Coupard (*Journ. de Méd. de Bruxelles*, 1888, lxxxvi., p. 147).

I should add that some writers have described **reflex nervous troubles** as the result of adenoids in infancy. Amongst these are convulsions, tetany, laryngismus, and vomiting. A good example of the first of these has been described by Dun.* A baby five months old had had convulsions for three months, the attacks being at first infrequent—once or twice a week—but had increased later on in spite of treatment until they numbered four or five each day. No cause for the convulsions could be ascertained, but there had been occasional slight nasal discharge, and the baby snored when laid on his back. The naso-pharynx was scraped, and the convulsions straightway ceased. That is a very striking case which it will be well for you to bear in mind.

So much for the occurrence of adenoids in young infants. They may lead to symptoms which it may be difficult to diagnose from those due to other medical diseases, and they are symptoms which may lead to serious impairment of health.

ADENOIDS IN OLDER CHILDREN.

Now, when we pass to older children we find the condition is much easier to study, because in their case you can make certain of the existence of adenoids by digital examination. I need hardly remind you of the enormous frequency of adenoids in children up to the age of puberty. After that age they tend to disappear spontaneously. But children at the period of the second dentition suffer from adenoid growths with great frequency, and the danger is lest one should take an exaggerated view of the presence of these growths. Some of you who have attended my out-patients may think I am inclined to overestimate the importance of adenoids in children and the part which they play in medical diseases, and after the recent pronouncement of such an eminent specialist as Sir Felix Semon, perhaps it requires some little courage to maintain that adenoids produce remote or medical effects at all. Yet I think no one can see much of disease in children without being convinced of the frequency of such a condition, and the fact that it seems to stand in causal relationship to certain remote symptoms in many instances. I can only advance the histories of certain cases in proof of this statement.

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* *Brit. Med. Journ.*, 1903, ii., p. 590.

Let us consider, first of all, the relationship of adenoids in older children to *diseases of the respiratory system*, because it is upon the respiratory system that the consequences of adenoids most directly fall. Now, the first respiratory disease which it is important you should recognize as a common consequence of adenoids is simple **bronchitis.** You will frequently have children brought to you with severe cough, and on examining the lungs you will find a few moist sounds at the bases. Such children will be found to be mouth-breathers and snorers at night. It is not difficult to see how adenoids lead to bronchitis. What is the normal function of the nose? It has been calculated that the nose yields up to the inspired air as much as two quarts of water in twenty-four hours ;* it adds that quantity of water vapour to the air which we inspire besides warming it up to the body temperature. It has also been calculated by Sir St. Clair Thomson and Dr. Hewlett that 1,500 micro-organisms are inspired every hour, and that sometimes this quantity is enormously increased. Yet when you come to examine the mucous membrane of the trachea you find that it is practically sterile—that is to say, the micro-organisms do not get beyond the outer passages. Thus it will be seen that the function of the nose is to moisten the air and to sterilize it, and if the nose is obstructed, as it is in marked adenoids, these two functions are imperfectly performed, and the child is breathing an unduly dry air, which is, moreover, insufficiently sterilized, and the consequence of that may be that the lining membrane of the tubes gets irritated, and the child develops bronchitis. If you are going to treat a case of bronchitis of that sort successfully you must first see that the upper air-passages are rendered patent.

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* Mayo Collier, *Journal of Laryngology*, 1901, xvi., p. 21.

Another respiratory condition definitely associated with adenoids in certain cases is **asthma.** I recognize that I am here on more dangerous ground, because asthma is one of those reflex neuroses the occurrence of which is apt to be exaggerated. I shall have to mention one or two reflex neuroses which arise not uncommonly in patients who are the subject of blockage of the nose or naso-pharynx, and one of those I certainly think is asthma. Take these two cases. A girl, aged eight years, was brought to me not long ago with the complaint that she had suffered from asthma since infancy. On examination the chest was found to be flat and badly developed, and there were a few moist sounds at the base of each lung, as well as an extreme development of adenoids in the naso-pharynx. The adenoids were removed and no other treatment was adopted, and four months after the operation it was reported that she was perfectly free from the attacks. Another patient was a boy of six years and three months, who, since three and a half years of age, had been treated for asthma, but without any real benefit. The mother stated that he had a severe attack of nocturnal dyspnœa lasting for several hours occurring once a week. He had excessive enlargement of the tonsils and a large mass of adenoids. He also was operated upon without the adoption of other treatment. Three months later the mother reported that he was almost free from attacks except when he had a cold in his head. Without mentioning other cases in which there seemed to be a definite relationship between adenoids and asthma, those that I have referred to are quite conclusive. I admit there are cases of asthma and adenoids in which removal of the adenoids does not seem to cure—that is to say, if you find adenoids and asthma coexisting you cannot promise that removal of the adenoids will invariably cure the asthma. I had a very interesting case illustrating that point. It was that of a child five or six years of age who suffered from asthma. He lived in the country, and had suffered practically all his life from the disease. Unquestionably he also had a large mass of adenoids. He was brought to the hospital here, and when I saw him I recognized the presence of these adenoid growths, and I advised that they should be removed. This was done, and he remained in London several weeks. During that time he was practically free from attacks. Of course, I naturally concluded that the operation had cured the asthma, and he went back to his home in the country. I followed the case up, and I found that immediately he got back there the attacks of asthma recurred. This, therefore, seemed to be one of those cases in which the asthma only came on in certain localities, the child having been born in a locality which produced it in him. In London he remained free from attacks. These cases are not infrequent, and you may be easily deceived by them. The mere presence of adenoids does not insure that removal of them will cure the accompanying asthma.

The next system which may be affected by the presence of adenoids is the *alimentary*. Those of you who have seen much of children's diseases, especially in the out-patient department here, must have been struck by the fact that perhaps the majority of the cases of adenoids which one sees in children after the second dentition occur in those who are dyspeptic, and in those who suffer from that particular form of **dyspepsia** which I described in an earlier lecture (Lecture XIII.). It is difficult to be sure of what the relationship is between adenoids and dyspepsia. Some people go so far as to say that the constant swallowing into the stomach of the mucus secreted by the adenoids maintains a condition of dyspepsia. The only way of settling the matter would be to treat some of those cases by putting the naso-pharynx into a healthy condition, and observing what the effect was upon the stomach. That I have not done, but I hope to carry out the treatment on these lines in other cases, with the view of seeing what the relation is between the adenoids on the one hand, and the dyspepsia on the other. But certainly in these cases it is extremely important to recognize the existence of adenoids because of the very frequent mistake which is made in regarding them as cases of pulmonary tuberculosis.

The next system which may be involved in cases in which adenoids are present is the *nervous system*, and we have a large number of cases of nervous disorders associated with, and I think in many cases causally associated with, adenoids in the naso-pharynx.

To the first group belong cases of general **mental dulness** or *hebetude*. That is a condition—the name 'aprosexia' has been applied to it—which implies inability to concentrate the attention properly. If you go into any of the special classes which are formed in the London Elementary Schools for backward children, you can pick out at once a large number of cases which suffer from adenoids. The way in which adenoids may produce this mental dulness or backwardness has been much disputed. Some people have supposed that the adenoids interfere with the proper return of lymph from the brain. They believe that the lymphatics passing from the brain are blocked, and that therefore the brain is kept bathed in the products of its own metabolism, and in that way its proper action is interfered with. Perhaps that theory is rather far-fetched. A simpler view is that the mental dulness in these cases is due to inability to hear well, that the pupil never properly hears what the teacher says. Such children are not, strictly speaking, deaf; they hear noises, but they do not adequately distinguish words, particularly if they are at a little distance away, and hence they become backward in their class. Another theory seeking to explain an association between adenoids and this backward condition is that the brain is kept constantly in a state of partial asphyxia from the blood not being sufficiently oxygenated. Whatever the true cause is, there can be no doubt of the great frequency with which one finds this general mental dulness associated with adenoids. In all cases in which you are consulted about a child who is backward at school examine the naso-pharynx, and at the same time examine the ears. You will find in quite a large proportion of them that the interference with hearing is due to the presence of adenoids, and I can assure you, from my own experience, that many of them have been greatly improved by having the throat cleared out. I have that on the evidence of teachers in those special classes to which I have referred.

Another neurosis which may arise from the presence of adenoids is **night terrors.** It is not quite so easy in this case to explain the association, and the cases are complicated by the fact that in the majority of them there is that form of dyspepsia which I have already spoken of present as well. It is difficult to assign a proper share in producing the night terrors to the dyspepsia and to the adenoids respectively, but I have seen cases in which removal of the adenoids alone produced a great improvement in the night terrors. Rey has recorded* the case of a girl who was operated upon on five successive occasions for adenoids, and in every case this immediately relieved the night terrors, which only recurred with the recurrence of the adenoids themselves. That is a fairly conclusive case in regard to the association of the two. Certainly in the case of any child who comes under your observation with night terrors you should attend to the state of the naso-pharynx in addition to treating the general health. The proper way to do this is not to give nerve sedatives, such as bromides and chloral. The necessary thing is to attend to the digestive organs, and particularly to give the patients aperients.

Allied to night terrors is another neurosis, namely, **nocturnal incontinence of urine.** That is a condition which, beyond all doubt, is often *maintained* by the presence of adenoids, if, indeed, it is not actually caused by them. The first case of this sort that I saw was an instructive one, that of a boy in a Home in the east of London, who had suffered from nocturnal incontinence of urine nearly all his life. He was sent to me by the superintendent of the Home to see what could be done for him. I found he was a typical mouth breather, that he snored at night, and that he had a large mass of adenoids and enlarged tonsils. His throat was operated upon, and almost immediately the nocturnal incontinence ceased and did not recur. Similar cases have been recorded in large numbers by various other writers. The relation was first pointed out by Major in 1884, and was confirmed by other German writers subsequently; and Grönbech has recorded* 23 cases in which adenoids were operated upon, and of which twelve were fully cured, five were improved, two were somewhat benefited, three remained as they were, and one was lost sight of. Huber, in America, found† that out of 127 cases of adenoids sixty-one had incontinence, and of thirty-nine of them removal of the adenoids was followed in twenty-six by complete cure and in eleven by great improvement. So if you have a case of enuresis, certainly adenoids should be looked for, and if they are found they should be removed.

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* *Jahrbuch f. Kinderheilk.*, 1897, xlv., p. 316.

Amongst other and rarer forms of nervous disturbances to which adenoids may give rise one is **defect of speech.** I have seen several cases of adenoids associated with difficulty in speech. Usually these cases occur in young children, and in the majority of them there is reason to suppose that the adenoids have been congenital. A child of two years of age was brought to me because he was unable to use words at all. He seemed mentally quite bright, and apparently heard ordinary sounds well enough, and the only thing which could be found amiss was the presence of extensive adenoid growths. Operation produced partial improvement, but one found some time afterwards that the growths had recurred. That child has since been operated upon again, and it will be interesting to follow the case up and see what improvement of speech follows the operation. I do not know what the difficulty in the speech is due to in those cases; it may be that the child does not hear words spoken by other people sufficiently clearly to enable him to imitate them.

Another minor neurosis which may accompany adenoids is **headache.** It is usually frontal, and is probably due to interference with the proper ventilation of the air sinuses. Such headache is frequent in adenoids. Dr. Crowley‡ went through a large number of cases, amounting to some hundreds, which had been operated upon here, and in 55 per cent. of them he found there was a history of headache, which had been cured by operation in about 50 per cent. **Vomiting** has been recorded as one of the rarer forms of neurosis, particularly by Breton.* He describes recurrent vomiting in a child which was cured by removal of adenoids. I have not seen such a case, but the peculiar spasmodic cough which adenoids give rise to frequently ends in retching, if not in actual vomiting. **Torticollis** is another and rarer condition which has been reported to be cured by the removal of adenoids. Gillette records three cases.† A still rarer form of neurosis of which I have seen one example only, which seemed to be due to adenoids, is **ataxy** or *staggering*. I remember a child. aged three years, who was brought to me with the complaint that he was wasting, had attacks of nose bleeding, and was very backward of speech, and apparently did not hear well. The child had excessive adenoids, and the typical adenoid facies, mouth breathing, and other symptoms. I did not have the throat operated upon at once. Soon afterwards the child developed ataxy, and staggered about as if he were intoxicated. This ataxy became more marked after the lapse of a few days, until eventually he was hardly able to stand at all. He was then taken into the hospital, and on examining him more carefully one found that he had marked indrawing of the tympanic membranes. There was no sign of cerebellar disease—that is to say, no optic neuritis and no vomiting, neither was there any complaint of headache. The case was watched for some time, but did not improve. It was rather puzzling to know what the ataxy could be due to, and one thought it worth while to remove the adenoids on the theory that the indrawing of the tympanic membrane was affecting the semicircular canals. You know that a person with a large quantity of wax in the ear may suffer from acute staggering, and I thought it might be the same with this child. The adenoids were completely removed, and then the ears were inflated. Improvement began almost at once and continued steadily. The operation was performed nearly a year ago, and when I saw him quite recently he was in practically a perfect state of health, and I was told that there had been no tendency to ataxy since he left the hospital. I do not wish to claim that this was a case of ataxy due to adenoids, but I think it is very suggestive, as the improvement began immediately after operation.

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* *Arch. f. Laryngol. und Rhinol.*, 1895, ii., p. 214.

† Abstract in *Medical Review*, 1900, iii., p. 182.

‡ *Pediatrics*, 1897, iii., p. 385.

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* *Rev. mensuelle des Maladies de l'Enfance*, 18, p. 235.

† Abstract in *Pediatrics*, 1896, ii., p. 540.

Such, gentlemen, are some of the medical diseases which may be simulated or caused by adenoids, and the question is what degree of importance one should attach to the presence of adenoids in any given case. And here again I warn you against the dangers of exaggeration. I do not want to say for a moment that all those cases which I have mentioned were directly *caused* by adenoids, but they were all, as such cases very frequently are, *associated with* adenoids; and in many of them when you remove the adenoids the other symptoms disappear. It is perfectly true that you can, in the majority of cases, find a predisposing cause for the bronchitis, or night terrors, or whatever it is, as well, yet in many of them one can fairly regard post-nasal growths as an exciting cause, and if you remove the growths great improvement follows.

TREATMENT.

Into the technique of the operation for the removal of adenoids I do not propose to enter. But one may say a word as to **when operation is indicated.** I do not wish you to understand that in all cases such as I have described if you find adenoids you should proceed to removal at once. I think it is safer, unless the symptoms are extreme, to try simpler measures first. Mr. Arbuthnot Lane has recommended **respiratory exercises** in the treatment of these conditions, and has said that if one has recourse to them systematically and persistently operation is in many cases unnecessary. Of such a line of treatment I have had little experience, because in the out-patient department of a hospital it is almost impossible to get it carried out with sufficient thoroughness and persistency to afford a reasonable chance of success. In ordinary out-patient practice I think the best plan is to try, first of all, ordinary cleansing of the nose. Very often one finds that symptoms are not due merely to the adenoids, but to their producing an excessive secretion of mucus, which becomes dry and causes blocking. If you can remove that, the adenoids of themselves may not be sufficient to produce serious symptoms. One of the best things for this is the **nasal douche.** A solution of borax (1 drachm to the pint) is a mild and fairly good solvent of mucus, and this is injected up the nostril and allowed to run out by the mouth so as to wash out the nasal cavity. In many cases this has resulted in a large degree of improvement. In any case, if you intend to proceed to operation it is well to submit the patient to such treatment before the operation is undertaken. One is less likely to get sepsis afterwards if one has first taken the trouble to see that the nasal passages are reasonably clean.

With regard to the **operation** itself, you must be prepared occasionally for failure—that is to say, you must be prepared for the possible recurrence of symptoms. When first I directed my attention to this subject, I used to think that in many of the cases in which the operation seemed to fail the removal of the adenoids had been incompletely carried out. But now I think that is probably not correct. In many cases I am certain that the adenoids recurred, and I think one may go further and say that the younger the child the more likely such recurrence is to take place, and it is often well to prepare the parents for that, and to tell them that you may require to perform two or three operations before you get a satisfactory result. It was found by Dr. Crowley that of the cases operated upon here, quite a considerable proportion showed recurrence, although there was no reason to suppose that the operation had not been completely performed. So if one finds that the symptoms are not relieved by the first operation it is not fair to blame the surgeon and allege that he has not done his work properly.

Another cause for apparent failure from operation may be that the nasal passages are abnormal. There is nothing commoner than for adenoids to be associated with some other cause of blocking in the nose, such as enlargement of the inferior turbinated bones, or a disordered condition of the septum, and so on. And, of course, you cannot expect in these cases that mere removal of obstruction behind will result in much improvement so long as the anterior obstruction still exists. So in some of these cases it may be necessary to attend to the state of the anterior nares as well as to remove the adenoids.

Lastly, though in many cases operation may be expected to cure those cases by itself, you must never forget to **treat the underlying condition** which is so often present. Adenoids may certainly be regarded as a symptom of ill-health, and whilst treating the local disease, you must attend also to the general condition. These children frequently want tonics of different sorts, particularly iron, and they also require attention to the stomach. Many of them, too, are much benefited by a change of air. In other words, whilst you are attending to the exciting cause of their symptoms you must never forget to give attention also to the predisposing cause, which is usually a delicate condition in the child.

LECTURE XXX

SOME OF THE COMMONER SKIN DISEASES OF INFANCY AND CHILDHOOD

GENTLEMEN.—Diseases of the skin are, I am afraid, rather uninteresting, and in childhood, at all events, they are fortunately very rarely in any way dangerous to life or even to health; but as their treatment will undoubtedly cause you a good deal of trouble in practice, I thought it might be well to devote a lecture to them. I shall deal with the subject mainly from the point of view of treatment, for I can profess to none of the specialist's knowledge of the pathology of cutaneous diseases.

It has to be recognized at the outset that in children the skin is peculiarly apt to suffer from disease of one sort or another, and that for several reasons. In the first place, it is of a softer and more delicate texture than it is in the adult, and therefore more prone to suffer from the consequences of mechanical or chemical irritation. In the second place, owing to the relatively larger surface of the child and the important part which the skin plays in the regulation of temperature, it is subject to great fluctuations in its blood-supply, which must predispose it to suffer from inflammatory processes. Thirdly, the vessels of the skin are apparently under less efficient nervous control in early life than they are in the adult, so that diseases of neuro-vascular origin, such as urticaria, and the erythemata, and reflex congestions (the result of digestive disorders), occur more readily than in later life. Lastly, children are more subject to the attack of parasites and to the consequences of a lack of cleanliness than grown-up people, whilst their intimate contact with each other in school and at their games renders

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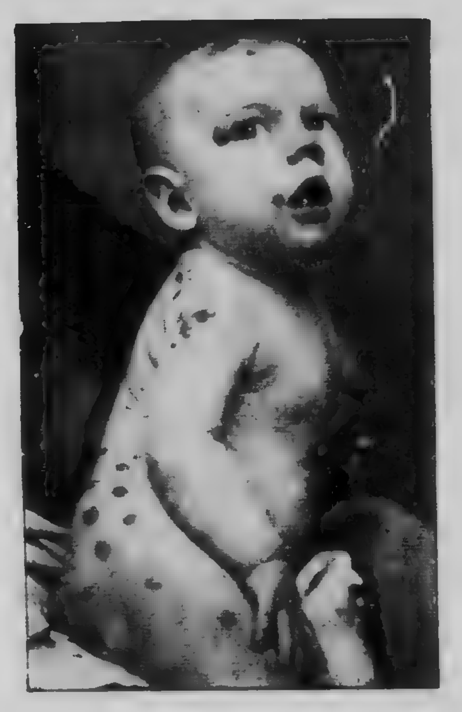

Fig. 69.

BROMIDE RASH

them specially prone to contagion. If you bear these points in mind it may help you to take measures to prevent the development of cutaneous diseases amongst any children who may be under your care.

When you are confronted with any case of skin eruption in a child, it is well before proceeding any further to ask yourself three questions : (1) Is it the eruption of one of the specific fevers ? (2) Is it the result of a drug ? (3) Is it due to an insect or parasite ?

SPECIFIC, DRUG, AND PARASITIC RASHES.

1. In answering the first of these questions the thermometer should keep you right. Be suspicious of any eruption which is accompanied by fever.

2. To dispose of the second question you must inquire what medicines, if any, the child has been taking. Now, almost any drug may, in a child which happens to have an idiosyncrasy to it, produce some sort of cutaneous eruption, but some are met with more frequently than others. Antipyrin, for instance, produces an erythema fairly often. A copaiba rash may very closely simulate measles or scarlatina, and I have even known it to be accompanied by some pyrexia ; but, fortunately, copaiba is not often administered to children. Commonest, perhaps, and certainly the most serious of all, is a bromide rash. In little babies a **bromide rash** presents quite distinctive features. It is not—as it is in the adult—pustular, but consists of curious fleshy papules, sometimes almost cauliflower-like, which are scattered discretely over the body, but with a special tendency to affect the head and face (Fig. 69). They are not inclined to suppurate, but pursue an indolent course, and when they disappear leave scars behind. The true cause of such an eruption is very apt to escape recognition.

3. As regards the rashes produced by insects and parasites, I would remind you not to mistake the results of the flea or other **bites** for purpura, as I have known done more than once. The presence of a darker puncture in the centre of each spot in the case of bites should prevent such an error. Mosquito or midge bites may produce rather puzzling

appearances, and, as I shall have occasion to point out further on, they may closely simulate an erythema nodosum. Scabies as it affects children I shall refer to later. It is characteristic of all these rashes produced by insects and parasites that they *itch*, and in a doubtful case this may be of diagnostic value.

INTERTRIGO.

Perhaps the simplest form of skin disease is that superficial form of dermatitis which we speak of as intertrigo. The appearance of this is too well known to all of you to need any special description, but, as its name implies, it is the result of a fretting or galling of the skin by some superficial irritation. Such irritation may be brought about by the excreta, in which case extensive intertrigo may occur over the parts covered by the napkins, whence it may spread to the backs of the legs and the heels, from their being drawn up towards the buttocks during attacks of colic. It is also met with, usually in fat babies, from irritation by decomposition of the secretions of the skin wherever two cutaneous surfaces are in contact—as, for example, behind the ears, in the transverse folds of the neck, or, more rarely, in the axillæ.

The **diagnosis** of simple intertrigo is not usually a difficult matter, but it is not always easy to tell it when it is confined to the buttocks and perinæum from the rash of congenital syphilis. The fact, however, that it is limited to those parts which are covered by, or in contact with, the napkins, that there is no eruption on the palm or soles, that the eruption is bright red instead of being reddish-brown or 'coppery,' along with the absence of other evidences of congenital specific disease, should keep you right.

In the **treatment** of intertrigo cleanliness is of the first importance. The mildest soaps, however, should be used, and in some cases it may be advisable to dispense with their use altogether, and to use oatmeal or bran instead. An antiseptic dusting powder, of which there are now so many excellent forms on the market, should be applied wherever two skin surfaces touch, or they may be separated by a piece of linen spread with zinc ointment. The intertrigo which occurs behind the ear is apt to be very resistant to treatment, and it is often a good plan in such a case to paint over the affected area a solution of nitrate of silver in sweet spirits of nitre (16 grains to the ounce). This produces a little momentary smarting, but its application is soon followed by a drying up of the oozing surface, and the formation of a protecting pellicle, under which healing quickly takes place.

In treating the intertrigo which occurs under the napkins the first step is to deprive the excreta of their irritating properties. You will often find that the baby in such a case is being overfed, and some reduction in the amount of frequency of the feeds is necessary. The use of starchy foods, too, must be stopped, for undigested starch leads to the formation of acid stools, which are very irritating. If there be any looseness of the bowels, a few grains of aromatic chalk should be administered two or three times in the day. If the motions are curdy without being loose, small doses of rhubarb and soda may be given instead. Locally great care should be taken to keep the parts clean. The napkins should be changed as soon as they are soiled, and dusting powder or zinc ointment applied to the affected parts.

ECZEMA.

Closely allied to intertrigo, and, indeed, sometimes arising out of it, is eczema. The characteristic form in which this affection manifests itself in childhood is **eczema of the face and head.** Now, I think you will find that eczema specially tends to attack babies of a particular type. There is really quite an extraordinary resemblance between eczematous babies. Briefly I may say that they are nearly always well-nourished infants, with a fine, smooth, white skin, light blue eyes, and fair hair—quite an attractive type of baby, in fact—which makes their being the victims of this disfiguring complaint all the more pathetic. I suppose the meaning of it is that it is the very fineness of their skin which makes it subject to the form of catarrh we call eczema. The disease usually begins over the malar bones and spreads upwards over the scalp, and downwards over the rest of the face and upper part of the trunk. In its full-blown form it manifests itself by redness and oozing, with much local heat, so that when a moist dressing is removed you will often find it steaming hot. The serous fluid which exudes from the surface speedily dries into a yellowish cake or crust, which may cover the whole scalp and face, and from which the light blue eyes peer out with a clearness and brightness which is sharpened by contrast with the loathsome frame in which they are set.

I have indicated my belief that the predisposing **cause of this form of eczema** is the possession by the child of a type of skin which is peculiarly susceptible to irritation; but if you ask me what the exciting cause, the actual starter, of the eczema in these cases is, I must confess my ignorance. You will hear it attributed by doctors to some fault in the diet, and by mothers to 'teething.' As regards the former of these opinions, I can only say that my experience is not in harmony with it. In many—perhaps the majority—of such cases the infant is being fed at the breast, and is in every way a well-nourished child who has no digestive disturbance at all. If food does play any part in the process, I am convinced that it is more often the quantity than the quality of the diet which is at fault, and that if you make any alteration in the feeding, it should be in the direction of reducing the amount of the feeds. As regards the question of teething, I can only say that, whilst I do not believe that by itself teething often causes eczema, yet there can be no doubt that, pending the eruption of a tooth, an already existing eczema is apt to be aggravated, and to take on a more angry and inflammatory character. I have seen this happen too often in eczematous babies who have been under treatment in the wards to have any doubt on the subject. In the same way, any disorder of digestion may intensify an eczema temporarily, and the flushing of the face after meals which is so familiar a phenomenon in dyspepsia gives a key to the understanding of how this may be.

If the views I have expressed be correct, you will not expect much from general or **internal remedies** in the treatment of the eczema of childhood. By all means correct any error in feeding which you may detect ; if there be constipation, administer saline aperients, and if the motions are ill-digested and unhealthy, try rhubarb and soda. Such recommendations are obvious. There remain three drugs that are said to be useful. Citrate of potash is often administered in these cases under some vague impression that it is 'cooling' to the blood. I can only say that I have tried it fairly diligently, and am not sure that it exerts any beneficial effect. Guaiacum has been recommended by Dr. Eustace Smith. It is given in the form of the tincture in doses of 10 minims three times a day. It is said to take the 'fieriness' out of an eczema, but although I have often intended to try it I have never done so yet. Quinine is said to exert a favourable effect in some cases. It is given in doses of 1½ grains for every year of the child's age once a day. Euquinine—which is tasteless—is the best form in which to exhibit it.

When all is said and done, however, it is upon persistent **local treatment** that you must chiefly rely. The first step is to clear away all débris and get a fair field for your remedies. Crusts should be removed by the use of boracic starch poultices* or by soaking with oil. When softened by one of these means, they must all be picked off, so that a bare oozing surface is left. So long as the eczema continues to 'weep' there is no use in applying ointments, for they will not stick, and you must depend upon lotions. I use one of three—calamine, lead lotion, or black wash. By such means the inflammation can be kept at bay, and when it begins to subside, and the eczema assumes a more scaly form, you may start to use ointments, of which the best, I think, is one containing 5 grains each of resorcin and salicylic acid in an ounce of vaseline. If there be much tendency to pus formation, it may be better to use dilute white precipitate ointment first. If stronger remedies cannot be borne, ½ drachm of oleate of zinc in an ounce of cold cream forms a good soothing application. The ointment should be applied on strips of linen covered with a mask provided with eye-holes, and fitting tightly over the scalp. In order to prevent attempts at scratching, it may be necessary at this or any stage of an eczema to apply light pasteboard splints along the flexor aspects of the arms, so that the hands cannot reach the head.

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* Prepared by mixing 1 teaspoonful of boric acid and 1 tablespoonful of cold-water starch to a paste with cold water, then adding 1 pint of boiling water. The paste should be spread on cotton, covered with muslin, and changed often.

Care must be taken to protect the face from cold winds or sun when the child goes out. A red veil is useful for these purposes. The skin should not be washed with soap, but bathed with water containing 1 drachm of borax to the gallon.

No matter how careful and thorough your treatment may be, you must be prepared for **relapses,** and, what is more, you must prepare the mother for them too. I always make it clear when undertaking the treatment of a case of eczema in an infant that one must not look for a certain cure, but that the condition is likely to persist with ups and downs for a long time, possibly until all the teeth are cut. You may safely promise, however, that it will get well in time, and that it will leave no bad effects or disfigurement behind.

SEBORRHŒIC ECZEMA.

The term 'seborrhœa' is a bad one—one of the worst in dermatology, in fact, which is saying a good deal—for it naturally suggests an increased production of sebum, which does not really take place. It is convenient, however, to retain the term 'seborrhœic' for that form of eczema met with on the scalp and forehead and behind the ears, and occasionally also on the upper part of the trunk, which is characterized, not by oozing, but by the production of greasy, yellowish flakes or scales, which may adhere into a brittle crust. If one removes the scales, the underlying skin is found not to be so congested as in ordinary eczema, but rather of a pinkish tint. Seborrhœic eczema is met with both in babies and in older children, and in the former it often forms a greasy, yellow layer on the top of the head, which the mother is afraid to remove for fear of injuring the fontanelle. Here, again, the first step in **treatment** is to remove the scales or crust. When this has been done (in the way already described for ordinary eczema) an ointment consisting of 10 grains each of salicylic acid and sulphur, with 1 drachm of oxide of zinc, in an ounce of vaseline, should be well rubbed in. You will find that it is more amenable to treatment than ordinary eczema.

IMPETIGO.

Impetigo contagiosa is a frequent affection of the skin in childhood. It is commonest in children of school age, and, as the name implies, is conveyed from one to the other by direct contact, being due, as you know, to local infection with pyogenic organisms. It most commonly affects the face, especially in the neighbourhood of the mouth, and you may easily recognize it by the yellow crusts, which have a curious look of having been stuck on artificially. Its **treatment** is as simple as it is satisfactory. You have merely to remove all the crusts in the same way as you do in eczema, and then to rub into the raw surface which is left a little weak antiseptic ointment. There is nothing better than white precipitate—5 grains to the ounce—of which a little should be well rubbed in *frequently* throughout the day. Under this plan the disease quickly disappears.

URTICARIA.

Nettle-rash, in one form or another, is one of the commonest of the minor ailments met with in the nursery, and everyone who has ever been stung by a nettle is familiar with the appearance which it presents in its simplest and uncomplicated form. Often, however, one finds that upon the top of the wheal there arises a papule which persists after the subsidence of the original eruption, and to this form the term **lichen urticatus** is applied. The term is justified, for the papules so formed have the typical flat, polished-looking apex characteristic of the papules of lichen. Sometimes little vesicles form on the top of them, and these may even go on to form pustules. Add to these some scattered wheals and the secondary lesions and infections which result from scratching, and you will readily understand that in its fully-developed form lichen urticatus may present rather a complicated picture. Now, there is another disease which may present an exactly similar picture, and that is **scabies.** For my own part, I confess my inability—in many cases, at least—to diagnose lichen urticatus from scabies merely by looking at it. For you must remember that scabies as it occurs in children has by no means the characteristic distribution which it manifests in the adult ; on the contrary, it is often quite a generalized eruption. In addition to this, both lichen urticatus and scabies cause great itching, especially at night, so that the mimicry of the one by the other is almost complete. How, then, are you to distinguish between them ? By attention to two points. Lichen urticatus is a disease of much longer duration than scabies : the life-history of the former covers months or years, that of the latter days or weeks. Secondly, scabies rarely affects but one child in a family, for before long another is almost sure to become infected. Should you still be in doubt, the therapeutic test will settle the point. Under a few sulphur baths (1 ounce of potassa sulphurata to 7 gallons of warm water), or the inunction of weak sulphur ointment (equal parts of sulphur and zinc ointments), or the application of equal parts of balsam of Peru and glycerine, scabies will quickly disappear ; lichen urticatus will not.

This leads me to speak of the **treatment of nettle-rash** in general, and at the outset I would remark that this is by no means an easy matter. Only too often you will find that the disease persists, with longer or shorter intermissions, for a long time, although, like eczema, it tends to disappear spontaneously about the end of the third year of life. I would advise you not to listen too readily to those who say that nettle-rash is always due to some disorder of digestion. Just as in the case of eczema, so here I have often found that there is really nothing to criticize in the food that is given or in the way in which it is digested, although I admit that in a few cases improvement will result from the use of the same general measures and internal remedies as were recommended in eczema. Aperients, rhubarb and soda, quinine, and ichthyol, all have their place, but, on the whole, you have to depend chiefly on local treatment. Nor is this at all sure to be successful ; often all that you can do is to relieve itching, and it is not always possible to effect even that. Avoidance of things which irritate the skin is of the first importance. Warm baths, for instance, may have to be interdicted, and in these cases at least the generally salutary rule of 'flannel next the skin' must be broken. Silk is the best material for the under-garments and night-dress, or, if silk be too expensive, linen. By attention to this point, I have sometimes cured cases in which all the ordinary remedies have been tried in vain. For the relief of itching many applications have been recommended. The following ointment is often efficacious :

β-naphthol	gr. xvi.
Zinci oxidi	ʒi.
Ung. simplicis	℥i.

This must be rubbed in all over the body at night. On the whole, however, there is nothing better than tar (equal parts of tar ointment and diachylon ointment), the only drawback to which is that it is dirty. A tar lotion (liq. picis carbonis, 1 drachm to a pint of water) is cleaner, but not, I think, quite so efficacious. A weak carbolic lotion (1 in 40) sometimes answers, but I am always rather chary of applying carbolic acid over large areas in young children.

It may also form an ingredient of other lotions, as, for example :

Acidi carbol.	ʒss.
Calamin. prep.	ʒiii.
Cret. prep.	ʒi.ss.
Glycerini	ʒi.ss.
Aquam	ad ℥vi.

To be painted on at night after washing with a menthol soap.

By the use of such means as these you may at least hope to alleviate the worst symptoms of the disease, but more than this it is often not in one's power to do. When all else has failed, however, never omit to try the effect of a change of air, which will often succeed—temporarily, at least—when local applications prove of no avail.

ERYTHEMATA.

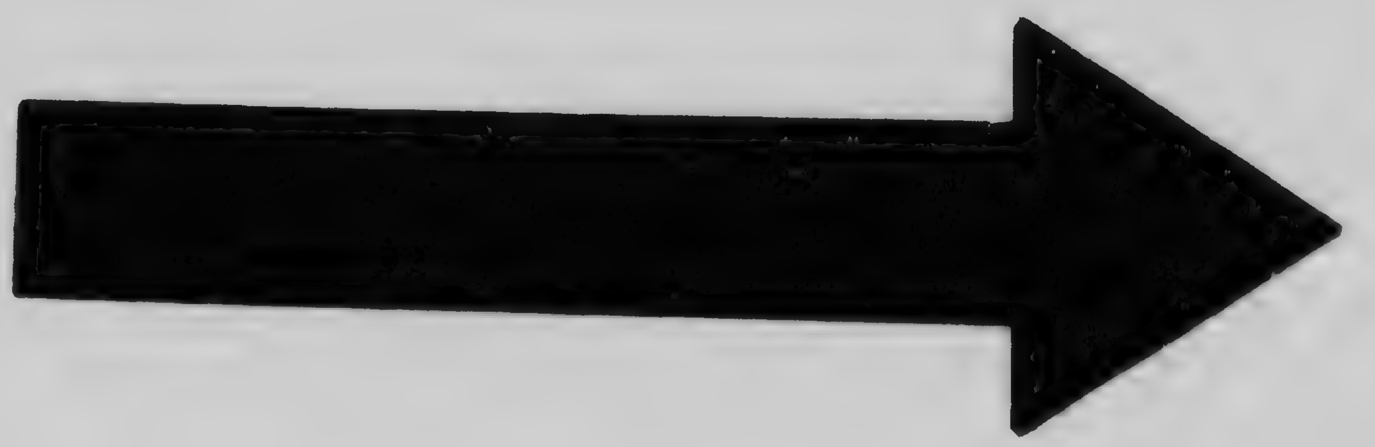
Putting aside the purely fugitive forms, erythemata tend to occur especially after the period of infancy, and you should always suspect an association of them with rheumatism. The commonest form is **erythema nodosum,** which occurs, as you know, chiefly on the legs, but occasionally on the arms as well, in the form of roundish, raised, and slightly tender blotches of a purplish colour, passing as they fade away through all the colours of a bruise. The only thing for which you may mistake it is the eruption which results from the bites of mosquitoes (met with even in this country), which may simulate a local outbreak of erythema nodosum very closely indeed. If you are aware of this source of fallacy, however, you are not likely to be misled by it.

The other form of erythema (**rheumatic erythema,** or peliosis rheumatica) is met with in the neighbourhood of a joint (which is usually rather swollen and tender), and forms a large area, dusky red and rather hot, with more or less hæmorrhagic exudation. It is much rarer than erythema nodosum, and is accompanied by more constitutional disturbance. In the **treatment** of both forms the only thing to be done is to put the child in bed. If you do this, the eruption very quickly fades. Salicylates may be given, but more as a preventive of other rheumatic manifestations than for the erythema itself, for I doubt very much whether the skin eruption is in any way affected by their administration.

SWEAT RASHES.

Rashes from excessive sweating are often met with in babies who are rather overclad, and especially, of course, in hot weather. I think their commonest distribution is over the base of the neck and upper part of the front of the chest. They consist of tiny vesicles (sudamina), often upon a basis of intertrigo. Sometimes they go on to form minute pustules, and when inflamed the eruption is spoken of by some writers as 'strophulus'; but as this term is also used by some to mean lichen urticatus, it is ambiguous, and is best avoided. The **treatment** of a sweat rash is the same as the local treatment of intertrigo.

Of psoriasis, which is certainly a fairly common skin disease in children, I do not propose to speak, for its management differs in no respect from that of the same disease as met with in adults. Nor shall I say anything of ringworm or alopecia, for I have nothing to add to what you will already find on these subjects in any text-book.



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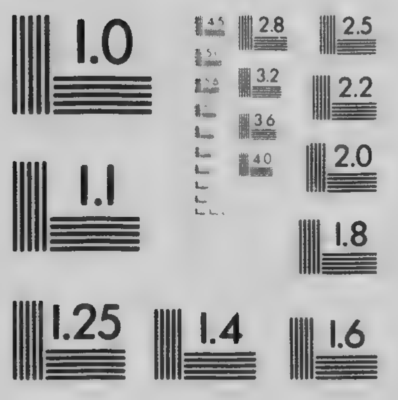

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